# THREE GENETICALLY INFORMED SOCIOLOGICAL STUDIES ON DATA QUALITY CONTROL, DELINQUENCY, AND RELIGIOSITY

Yi Li

A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Sociology.

Chapel Hill
2015

Approved by:

Guang Guo

Ted Mouw

François Nielsen

Michael Shanahan

Yang Yang

# ABSTRACT

Yi Li: Three Genetically Informed Sociological Studies on Data Quality Control, Delinquency, and Religiosity
(Under the direction of Guang Guo)

In this dissertation, I use molecular genetic data to conduct three sociological studies. In the first study, genetic information is used to improve the quality of self-reported familial relationships such as twins and full siblings, race, and gender. In the second study, I examine whether marriage moderates genetic effects for delinquency. In the third study, I examine whether risk and genetic risk can explain the gender gap in religiosity.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

# CHAPTER 1 –DATA QUALITY CONTROL IN SOCIAL SURVEYS USING GENETIC INFORMATION

## Introduction

The past decade has seen a number of large-scale social surveys collect molecular genetic data, for example, the National Longitudinal Study of Adolescent Health (Harris 2013), the Fragile Families Study (Reichman, Teitler, Garfinkel, and McLanahan 2001), and the Health and Retirement Study (Crimmins, Guyer, Langa, Ofstedal, Wallace, and Weir 2009). Recently, studies that incorporate genetic measurement have started to appear in leading journals (e.g., Caspi, McClay, Moffitt, Mill, Martin, Craig, Taylor, and Poulton 2002; Domingue, Fletcher, Conley, and Boardman 2014; Mitchell, Notterman, Brooks-Gunn, Hobcraft, Garfinkel, Jaeger, Kotenko, and McLanahan 2011; Shanahan, Vaisey, Erickson, and Smolen 2008). Scholars in social and biological sciences are also calling for further integration of social, behavioral, and genetic research. In an effort toward this end, a national committee issued a report emphasizing the importance of research on interactions between social and genetic environmental factors and recommending development of efficient study designs and rigorous models to test gene and environment interactions over time (Hernandez and Blazer 2006).

Genetic data not only enrich theoretical frameworks and empirical models in the social sciences, but also may offer promise with respect to data quality control in surveys. This article explores the role of genetic information in checking, repairing, and recovering self-reported variables. For example, when a self-reported variable has a missing value or is misreported, DNA data can provide additional information to detect and settle the issues in some circumstances. Proportion identical by descent (IBD) score (a measure of genetic relationships), bio-ancestry score (a measure of ancestral population memberships), and sex chromosomal information are all types of genetic information that may be used to improve self-reported variables such as sibling type, race, and sex. Improving data quality can be viewed a useful by-product of the availability of DNA data in social surveys.

In this article, we first describe the data sources and measures. Then we present the use of the proportion IBD score to check self-reported sibling type and to correct a more general issue—flawed data. Next, we show that missing race and discrepancies among different measures of self-reported race may in some cases be repaired by bio-ancestry score. Next, we demonstrate how to use sex chromosomal information to check self-reported sex. Lastly, we discuss implications of the findings and limitations.

## Data and Measures

### Data

Two data sources are employed in this article. The first is the College Roommate Study (ROOM) (Guo, Hardie, Owen, Daw, Fu, Lee, Lucas, McKendry-Smith, and Duncan 2009). ROOM was conducted at a public university in the spring semester of 2008 and sampled freshmen, sophomores, and juniors. Its main goal was to investigate peer influence on health-related behaviors such as drinking and smoking. Every year freshmen and returning students apply for housing. Students who do not request a specific roommate and do not request to participate in a themed housing program (e.g., substance free, foreign languages, health sciences, and global business) are eligible for randomly assigned roommates. These students were the targeted population. ROOM was a two-stage survey. The first stage was an online survey. Emails with the description of the survey and instructions on how to complete the survey were sent to students multiple times. Students were asked to answer questions about their health behaviors and socioeconomic backgrounds. The second stage involved collecting saliva from the students. Some 2,080 students (78.7 percent of those who completed the online survey) gave saliva to generate DNA data. Students who did not give saliva, lived off campus, or were studying abroad during the semester were eliminated for a final sample of 2,065 students.

The second data source is the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a longitudinal study that surveyed a representative sample of U.S. adolescents in grades 7-12 during the 1994-95 school year (Harris 2013). About 90,000 adolescents from 134 schools were surveyed. A subset of respondents, about 20,000, were interviewed at home and followed in subsequent waves. Interviewers asked for a wide range of information such as social backgrounds and physical health during interviews. Respondents were followed in 1996 (Wave II), 2001-02 (Wave III) and 2008-09 (Wave IV). The current article draws on data collected from a subsample of full siblings and twins at Wave III. At Wave I, a sibling subsample composed of full siblings, twins, half siblings, and unrelated pairs, was drawn. At Wave III a total of 2,612 individuals in the sibling sample gave saliva. The final sample consists of 2,281 individuals whose DNA was successfully genotyped.

Measures of DNA

In ROOM participants provided saliva in an Orangene DNA collection kit (DNA Genotek; Ottawa, Ontario, Canada). DNA was extracted from 2 mls of saliva, with a median DNA yield of 27.3 µg. DNA was plated for Illumina genotyping at 30 ul at >50 ng/ul. In ROOM, we designed an Illumina GoldenGate assay for 384 candidate genetic variants—single nucleotide polymorphisms (SNPs)—in 28 genes (for technical details see Guo et al. 2009) and 350 SNPs in 28 genes were successfully genotyped. In Add Health, DNA gathered at Wave III in 2002 was isolated from buccal cells at the Institute of Behavior Genetics at the University of Colorado, Boulder. The average yield of DNA was 58 ± 1 µg. The genotype data used in this analysis were based on an Illumina GoldenGate assay. The GoldenGate array targeted 1,536 SNPs. A total of 1,140 SNPs in 130 genes were successfully genotyped. The 28 genes and 130 genes were selected mainly because of their implications in risky behaviors such as aggression, alcohol use, smoking, and illegal drug use.

Measures of Proportion Identical by Descent Score and Self-reported Sibling Type

Identical by descent (IBD) refers to alleles that are the same in pairs of persons, and are inherited from a common ancestor (Malécot 1948; Wright 1917). Proportion IBD score is a measure of how much a pair of persons is genetically linked to the same ancestor on a scale of 0 to 1. For example, the proportion IBD of identical or Monozygotic (MZ) twins is 1 because they share the same genome from the same parents. Fraternal or Dizygotic (DZ) twins and full siblings on average share 50 percent of the genome, for a proportion IBD score of 0.5 on average.

PLINK (Purcell, Neale, Todd-Brown, Thomas, Ferreira, David Benderb, Sklar, Bakker, Daly, and Sham 2007), a whole genome association software program, estimates proportion IBD scores for all possible pairs in a dataset. PLINK uses the following formula to calculate proportion IBD scores:

$$P_1(IBD = 2) + 0.5 \times P_2(IBD = 1)$$

where $P_1$ is the proportion of loci possessing two identical alleles and $P_2$ is the proportion of the same loci possessing only one identical allele. The higher the score is, the more genome the pair shares. The value is 1 if two individuals have the same alleles, and 0 if they have no alleles in common.

Of the two surveys, only Add Health collected information on familial relationships. Respondents who had household members in the study were asked if they were twins or siblings. The types of pairs were further divided into MZ twins, DZ twins and full siblings. All mixed-sex twin pairs were classified as dizygotic. Same-sex twins were determined to be MZ or DZ based on self-reported confusability of appearance at Wave I (i.e., look like two peas in a pod as young children; and three other items, confused by strangers, teachers, or family members). When self-reported data on appearance was missing, the sibling type was determined based on the mother's report of confusability of appearance. Some twin pairs were classified as uncertain status because the self-reported zygosity differs from the classification based on confusability of appearance. Add Health also collected survey information on parent-child pairs, but no genetic data on this type of pairs is available.

Of 2,281 individuals in the Add Health sample, there are 884 pairs, 1,768 individuals, in which both members have genotype data to obtain a proportion IBD score. Of the remaining 513 individuals, proportion IBD scores cannot be estimated for 501 because their siblings do not have genotype data. For the final 12, the scores cannot be estimated because information on whether the individuals are members of a sibling pair is not available.

Measures of Bio-ancestry Score and Self-reported Race

A panel of Ancestral Informative Markers (AIMs) is used to estimate bio-ancestry score (a measure of the genetic composition of geographic ancestral populations). AIMs are sets of genetic polymorphisms, whose allele frequencies differ significantly across populations (Frudakis, Venkateswarlu, Thomas, Gaskin, Ginjupalli, Gunturi, Ponnuswamy, Natarajan, and Nachimuthu 2003; Parra, Marcini, Akey, Martinson, Batzer, Cooper, Forrester, Allison, Deka, Ferrell, and Shriver 1998; Shriver, Smith, Jin, Marcini, Akey, Deka, and Ferrell 1997).

Both Add Health and ROOM targeted 186 SNPs as AIMs , which were developed at David Goldman's Laboratory of Neurogenetics at NIAAA for the purpose of detecting broad, continental African, East Asian, and European populations (Enoch, Shen, Xu, Hodgkinson, and Goldman 2006). Of the 186 AIMS, 162 were successfully genotyped in ROOM, and 121 were successfully genotyped in Add Health. The AIMs are sufficient to distinguish African, East Asian and European populations, but are less effective in determining more specific sub-populations within the three populations (e.g., Chinese population within the East Asian population). Rosenberg and colleagues (2002) reported that 100 to 160 SNPs are sufficient when the sample size is about 1,000. In other studies similar conclusions were made (Halder, Shriver, Thomas, Fernandez, and Frudakis 2008; Smith, Lautenberger, Shin, Chretien, Shrestha, Gilbert, and O'Brien 2001; Yang, Li, Criswell, Gregersen, Alarcon-Riquelme, Kittles, Shigeta, Silva, Patel, Belmont, and Seldin 2005).

Bio-ancestry scores are estimated based on the AIM panel using STRUCTURE cluster analysis (Pritchard, Stephens, and Donnelly 2000). STRUCTURE is a software package that investigates an individual’s ancestral populations, independent of self-reported race. Assuming a mixture of multiple populations comprises the genome of each individual, the STRUCTURE analysis estimates the relative contributions of these populations to the individual. A K value needs to be set in the STRUCTURE analysis to indicate the hypothesized number of ancestral populations. The K contributions from K ancestry populations for each individual sum to 1.

Because our panel of AIMs was designed to differentiate continental populations of Africans, East Asians, and Europeans, we set K=3. In other words, individuals in the samples are assumed to have three bio-ancestry scores—African, East Asian, and European. The sum of the three scores is 1. To get more precise estimates, the STRUCTURE analysis is run 20 times and bio-ancestry scores are averaged on the 20 sets of results. All pair-wise symmetric similarity coefficients (SSC), which reflect the similarity of two sets of population structure estimates, are greater than 0.995. Our approach is similar to those in other studies of genetic population structure (Friedlaender, Friedlaender, Reed, Kidd, Kidd, Chambers, Lea, Loo, Koki, Hodgson, Merriwether, and Weber 2008; Wang, Lewis, Jakobsson, Ramachandran, Ray, Bedoya, Rojas, Parra, Molina, Gallo, Mazzotti, Poletti, Hill, Hurtado, Labuda, Klitz, Barrantes, Bortolini, Salzano, Petzl-Erler, Tsuneto, Llop, Rothhammer, Excoffier, Feldman, Rosenberg, and Ruiz-Linare 2007).

ROOM data includes two indicators of self-reported race and ethnicity. The first is from a housing application that incoming freshmen submitted to university housing when requesting a dorm room. The university housing form only allowed students to self-identify as a member of one of six racial/ethnic groups: “White,” “Black,” “Hispanic,” “Asian and Pacific Islander,” “Native Indian” and “Other.” The second indicator comes from the online survey, which allowed respondents to mark one or more races. ROOM first asked respondents whether they were of Hispanic origin and then asked their race. Respondents can select all that apply from the five options: “White,” “Black,” “Asian and Pacific Islander,” “American Indian or Alaska Native” and “Other.”

Add Health collected multiple measures of race and ethnicity at Waves I and III, but not at Wave II. The Add Health race/ethnicity questionnaire first asked respondents to indicate whether they were of Hispanic origin. Next, respondents were allowed to identify with more than one racial group from “White,” “Black,” “Asian and Pacific Islander,” “American Indian or Alaska Native” and “Other.” In addition, a respondent was asked to indicate which single-race category best describes her if this respondent marked more than one race. During the interview at Wave III, interviewers were instructed to record what they thought to be the respondent’s single-best race (independent of what the respondent said). The categories available for interviewers were “White,” “Black,” “American Indian or Alaska Native,” “Asian or Pacific Islander” and “Other.” “Hispanic” was not an option.

## Proportion Identical By Descent Score, Sibling Type and Flawed Data

### Proportion Identical by Descent Score, Self-reported Sibling Type, and Identifying “Misreported” and Unreported Sibling Type

Table 1 presents proportion IBD score by sibling type in Add Health. Proportion IBD scores are estimated in PLINK using all of the available 1,140 SNPs. Results show that the mean proportion IBD scores for DZ twins and full siblings are both consistent with the expected value—0.5. However, the mean proportion IBD score for MZ twins is not 1.00, but 0.92. This is due to six pairs previously identified as MZ twins based on reported confusability. Proportion IBD scores of the six pairs range from 0.46 to 0.58. Thus, the six pairs of twins are DZ twins.

TABLE 1.1
Proportion IBD Score by Reported Sibling Pair Type, Add Health

| | Proportion IBD Score | | |
|---|---|---|---|
| Reported Sibling Pair Type | Mean | Std | N of Pairs |
| Full Sibling | 0.52 | 0.11 | 633 |
| DZ Twins | 0.51 | 0.13 | 212 |
| MZ Twins | 0.92[a] | 0.18 | 34 |
| Undetermined Twins | 0.48 | 0.35 | 5 |

[a] Proportion IBD scores of six pairs are not 1.00. The six pairs were determined to be MZ twins by self-reported confusability. Proportion IBD scores suggest the six pairs are DZ twins.

Proportion IBD score may also be used to help identify unreported or unknown sibling type. Five undermined twin pairs in Table 1 are classified according to the proportion IBD scores. One of five pairs is MZ twins with a proportion IBD score of 1.00. Four pairs are DZ twins with proportion IBD scores ranging from 0.04 to 0.59. Add Health classified some twin pairs of uncertain status on the basis of four variable number of tandem repeats (VNTR) loci and three tetra-nucleotide microsatellite loci. Our proportion IBD scores are consistent with results from the seven genetic markers.

<u>Proportion Identical by Descent Score and Detecting Flawed Data</u>

A more general usage of proportion IBD score is to detect flawed data such as misrepresented/falsified respondents. Sometimes respondents participate in a survey for a second time under another person's name for incentives such as pay. It is also possible that due to errors in handling or data recording an individual's social and biological information may be recorded more than once. In these cases, proportion IBD score could help identify problematic pairs and correct them.

Case in point, in ROOM two participants were found to have a proportion IBD score of 1, meaning their DNA samples are identical. In the two identical DNA samples, 6 of 19 SNPs on X chromosomes are heterozygous. If SNPs on X chromosomes are heterozygous, it is very likely that the genetic sample belongs to a female. The genetic evidence, therefore, suggests that a female participant provided a DNA sample twice. Of the two participants, one reported as female in the online survey and the roommate was also female, whereas the other participant reported as male and the roommate was also male. Campus housing policies do not permit coed roommates, so the roommate sex confirms the reported sex of these respondents. Also, the female gave saliva first, and we would assume that if a person were providing a DNA sample in another person's name he or she would do it after providing his or her own DNA sample. Thus we concluded that the female may have given saliva twice for the monetary incentive. We decided not to link the male to this genetic sample. We also checked the female participant's online survey and found the answers to be within the reasonable range and no outliers that warranted exclusion. So we kept the survey responses.

**Bio-ancestry Score and Self-reported Race**

Bio-ancestry Score and Self-reported Race

Tables 2 and 3 present average bio-ancestry scores for self-reported race in ROOM and Add Health respectively. For Add Health, results of Wave I and III are very close. We only present those based on Wave I self-reported race. Both Tables 2 and 3 show that bio-ancestry scores are closely associated with self-reported race. For example, in ROOM among self-reported "Black" in response to a multi-race question the average African ancestry is 0.89, and is 0.87 among blacks in response to a single-race question; the corresponding numbers in Add Health are 0.93 and 0.91 respectively.

TABLE 1.2
Distribution of Average Bio-ancestry Score for Self-reported Race, ROOM

| Self-reported Race | Average Bio-ancestry Score | | | Sample Size |
|---|---|---|---|---|
| | European | African | East Asian | |
| Multiracial-allowed Race (Online Race) | | | | |
| White | 0.98 | 0.01 | 0.01 | 1,406 |
| Black | 0.09 | 0.89 | 0.02 | 281 |
| East Asian | 0.04 | 0.00 | 0.96 | 86 |
| South Asian | 0.68 | 0.05 | 0.26 | 41 |
| American Indian | 0.64 | 0.16 | 0.20 | 4 |
| Multiracial | 0.61 | 0.26 | 0.13 | 172 |
| Other | 0.69 | 0.15 | 0.15 | 62 |
| Missing | 0.81 | 0.17 | 0.01 | 13 |
| Total | | | | 2,065 |
| | | | | |
| Single Race (Housing Application Race) | | | | |
| White | 0.98 | 0.01 | 0.01 | 1,331 |
| Black | 0.11 | 0.87 | 0.02 | 338 |
| Asian | 0.29 | 0.02 | 0.69 | 151 |
| American Indian | 0.80 | 0.09 | 0.11 | 18 |
| Hispanic | 0.82 | 0.07 | 0.11 | 132 |
| Other | 0.84 | 0.09 | 0.07 | 94 |
| N/A | 0.98 | 0.02 | 0.01 | 1 |
| Total | 0.77 | 0.16 | 0.07 | 2,065 |

TABLE 1.3
Distribution of Average Bio-ancestry Score for Self-reported Race, Add Health

| Self-reported Race | Average Bio-ancestry Score European | African | East Asian | Sample Size |
|---|---|---|---|---|
| Multiracial-allowed Race, Wave I | | | | |
| White | 0.95 | 0.02 | 0.03 | 1,437 |
| Black | 0.06 | 0.93 | 0.01 | 381 |
| Asian | 0.07 | 0.01 | 0.92 | 160 |
| American Indian | 0.63 | 0.05 | 0.32 | 19 |
| Multiracial | 0.67 | 0.21 | 0.12 | 92 |
| Other | 0.61 | 0.10 | 0.29 | 179 |
| Missing | 0.53 | 0.39 | 0.08 | 13 |
| Total | | | | 2,281 |
| | | | | |
| Single Best Race, Wave I | | | | |
| White | 0.95 | 0.02 | 0.03 | 1,486 |
| Black | 0.07 | 0.91 | 0.02 | 406 |
| Asian | 0.10 | 0.01 | 0.89 | 171 |
| American Indian | 0.66 | 0.06 | 0.28 | 24 |
| Other | 0.61 | 0.10 | 0.28 | 180 |
| Missing | 0.53 | 0.39 | 0.08 | 14 |
| Total | | | | 2,281 |

When Self-reported Race Has Missing Values

Because bio-ancestry scores are closely associated with self-reported race, as shown in Tables 2 and 3, we propose that bio-ancestry scores can serve as a potential tool for data checking and repairing when reported race data are missing. However, missing or "misreported" race may carry sociological meanings. Depending on the goals of specific research, data users make the decision of whether to use bio-ancestry score to "impute" or replace reported race variables.

Table 4 presents bio-ancestry scores and housing race for all participants whose online survey race is missing in ROOM. The top panel of Table 4 shows that seven individuals' bio-ancestry scores are consistent with the race reported on their housing applications and the bottom panel shows that six individuals' bio-ancestry scores are not consistent with the housing application race. Note that in the bottom panel the six individuals' housing application races are not "White," "Black" or "East Asian." If the race information in the housing applications was not available, race of these 13 respondents would be treated as missing. With the bio-ancestry scores, however, each of the 13 respondents could be assigned to a non-missing race variable.

TABLE 1.4
Bio-ancestry Score and Housing Application Race for All Respondents Whose Online Survey Race is Missing, ROOM

| | Bio-ancestry Score | | | | |
|---|---|---|---|---|---|
| Pseudo ID | European | African | East Asian | Housing Application Race | Bio-ancestry Consistent with Housing Application Race? |
| 1001 | 0.992 | 0.003 | 0.005 | White | Yes |
| 1002 | 0.996 | 0.001 | 0.002 | White | Yes |
| 1004 | 0.996 | 0.003 | 0.002 | White | Yes |
| 1005 | 0.976 | 0.002 | 0.022 | White | Yes |
| 1006 | 0.992 | 0.002 | 0.005 | White | Yes |
| 1007 | 0.995 | 0.002 | 0.003 | White | Yes |
| 1003 | 0.178 | 0.820 | 0.002 | Black | Yes |
| | | | | | |
| 1008 | 0.845 | 0.136 | 0.019 | Hispanic | No |
| 1009 | 0.672 | 0.270 | 0.059 | Hispanic | No |
| 1010 | 0.985 | 0.012 | 0.003 | Hispanic | No |
| 1011 | 0.993 | 0.002 | 0.005 | Other | No |
| 1012 | 0.963 | 0.028 | 0.010 | Other | No |
| 1013 | 0.009 | 0.976 | 0.014 | Other | No |

Table 5 presents the bio-ancestry scores, self-reported race, and race observed by the interviewer for a representative subset of respondents whose self-reported race is missing in Add Health. Single-best race responses are not presented because they are consistent with multi-race responses for all these respondents. Table 5 is separated by the far right column into three panels. The top panel shows individuals whose bio-ancestry scores are consistent with the available race information, that is, self-reported race at another wave and interviewer-recorded race. In one of them (Pseudo ID is 1006), the interviewer-reported race is different from self-reported race. The respondent self-reported as “White” at Wave I but the interviewer marked the race to be “Asian” at Wave III. The European bio-ancestry score for this individual is 0.564, and Asian score is 0.312.

The middle panel shows individuals who are not classified as “White,” “Black” or

“East Asian.” Bio-ancestry scores are still helpful in these cases. For example, the respondent with Pseudo ID 1012 reported as “Other” at Wave I, but the interviewer-recorded race at Wave III is “White.” The bio-ancestry scores indicate that this difference is not surprising, because this respondent has an African ancestry of 0.159, East Asian ancestry of 0.470, and European ancestry of 0.371. The bottom panel of Table 5 shows two individuals whose bio-ancestry is not consistent with the survey information. For both respondents, self-reported race and interviewer-recorded race are “White,” while the highest ancestry scores are East Asian.

Suppose that bio-ancestry were not available in Add Health. Although missing race could be saved by other race variables in certain circumstances, there are places in which reported race variables are not sufficient to resolve the problem. For respondents with Pseudo IDs 1006 and 1008 to 1014 in Table 5, bio-ancestry scores are needed for insight into why these discrepancies between the race variables exist. Take the case of Pseudo ID 1011 as an example, with “Other” race reported at Wave I and interviewer-reported race “White” at Wave III, it is difficult to decide which of the two variables should be put in the missing race at Wave III. In addition, for the case in which all of the self-reported races are missing (Pseudo ID 1005 in Table 5), the missing might be “imputed” as “White” by the interviewer-reported race. But with this person’s bio-ancestry scores, a clearer picture is seen as this person possesses about 49.9 percent European ancestry and 37.5 percent East Asian ancestry.

TABLE 1.5
Bio-ancestry Score, Self-reported Race, and Interviewer-reported Race for a Representative Subset of Respondents whose Self-reported Race is Missing, Add Health

| Pseudo ID | Bio-ancestry Score | | | Self-reported Race | | Interviewer-reported Race | Bio-ancestry Consistent with Available Race? |
|---|---|---|---|---|---|---|---|
| | European | African | East Asian | Wave I | Wave III | Wave III | |
| 1001 | 0.978 | 0.008 | 0.014 | Missing | White | White | Yes |
| 1002 | 0.616 | 0.071 | 0.313 | Missing | White | White | Yes |
| 1003 | 0.050 | 0.902 | 0.048 | Missing | Black | Black | Yes |
| 1004 | 0.002 | 0.997 | 0.001 | Missing | Black | Black | Yes |
| 1005 | 0.499 | 0.126 | 0.375 | Missing | Missing | White | Yes |
| 1006 | 0.564 | 0.124 | 0.312 | White | Missing | Asian | Yes |
| 1007 | 0.995 | 0.003 | 0.002 | White | Missing | White | Yes |
| 1008 | 0.642 | 0.110 | 0.248 | American Indian | Missing | Asian | No |
| 1009 | 0.279 | 0.006 | 0.715 | American Indian | Missing | White | No |
| 1010 | 0.590 | 0.048 | 0.362 | American Indian | Missing | White | No |
| 1011 | 0.980 | 0.016 | 0.004 | Other | Missing | White | No |
| 1012 | 0.371 | 0.159 | 0.470 | Other | Missing | White | No |
| 1013 | 0.647 | 0.023 | 0.331 | Other | Missing | American Indian | No |
| 1014 | 0.554 | 0.444 | 0.003 | Multiracial | Multiracial | Black | No |
| 1015 | 0.386 | 0.172 | 0.442 | White | Missing | White | No |
| 1016 | 0.446 | 0.005 | 0.550 | White | Missing | White | No |

When Self-reported Race Differs across Different Measures or Waves

When self-reported race differs across various survey waves or measures, bio-ancestry score could offer additional information for reconciling such differences that might otherwise be considered reporting or recording errors.

Table 6 reports all the respondents who differ in bio-ancestry score, online race, or housing application race in ROOM. Yet, when comparing online survey race and housing application race, the category “Hispanic” is excluded because of the different options participants were given. In the online survey participants were first asked whether they were “Hispanic” and then to choose from “White,” “Black,” “Asian and Pacific Islanders,” “Native Indian” or “Other.” On the housing application, however, participants were asked to choose only one race/ethnicity from “Hispanic,” “White,” “Black,” “Asian and Pacific Islanders,” “Native Indian” and “Other.”

Table 6 reveals that there are three types of differences in ROOM. “Misreporting” is the first, and is illustrated in the top panel. In this context, “misreporting” means a large gap is found between self-classified race and bio-ancestry. For example, the first responder (Pseudo ID 1001) has a very high African bio-ancestry score of 0.994, and reported as “Black” on the housing application, but indicated “White” in the online survey. The middle panel shows mixed-race respondents. Researchers have reported that mixed-race individuals are more likely to report different races (e.g., Hitlin, Brown, and Elder 2006). Take the first respondent in the middle panel as an example (Pseudo ID 1003). This individual’s European ancestry score is 0.170 and African ancestry score is 0.817. With such a composition of bio-ancestry, this individual reported as “White” in online survey and “Black” on the housing application. The category for “White American Indian” is presented in the bottom panel of Table 6. All three respondents have very high European ancestry scores, ranging from 0.979

to 0.996, and they reported as "White" in the online survey. However, they all reported as "American Indian" on their housing applications. This finding may reflect "ethic re-identification" among American Indians (e.g., Eschbach 1993; Kelly and Nagel 2002).

TABLE 1.6
All Respondents Who Differ among Bio-ancestry Score, Online Survey Race and Housing Application Race, ROOM

| | Bio-ancestry Score | | | | |
|---|---|---|---|---|---|
| Pseudo ID | European | African | East Asian | Online Race | Housing Application Race |
| "Misreporting" [a] | | | | | |
| 1001 | 0.004 | 0.994 | 0.002 | White | Black |
| 1002 | 0.992 | 0.002 | 0.006 | Black | White |
| Mixed Race | | | | | |
| 1003 | 0.170 | 0.817 | 0.013 | White | Black |
| 1004 | 0.865 | 0.006 | 0.129 | White | Asian |
| 1005 | 0.363 | 0.003 | 0.635 | White | Other |
| 1006 | 0.520 | 0.476 | 0.004 | Black | Black |
| 1007 | 0.824 | 0.165 | 0.011 | Black | Black |
| 1008 | 0.608 | 0.386 | 0.006 | Multiracial | Black |
| 1009 | 0.539 | 0.458 | 0.002 | Multiracial | Black |
| 1010 | 0.612 | 0.381 | 0.007 | Multiracial | Black |
| 1011 | 0.590 | 0.406 | 0.004 | Multiracial | Black |
| 1012 | 0.448 | 0.003 | 0.549 | Multiracial | White |
| 1013 | 0.851 | 0.005 | 0.144 | Multiracial | Asian |
| 1014 | 0.516 | 0.004 | 0.48 | Multiracial | Asian |
| 1015 | 0.507 | 0.009 | 0.484 | Multiracial | Asian |
| 1016 | 0.533 | 0.003 | 0.464 | Multiracial | Asian |
| 1017 | 0.783 | 0.011 | 0.207 | Multiracial | Asian |
| 1018 | 0.585 | 0.008 | 0.407 | Multiracial | Asian |
| 1019 | 0.508 | 0.003 | 0.489 | Multiracial | Asian |
| 1020 | 0.573 | 0.005 | 0.421 | Multiracial | Asian |
| 1021 | 0.762 | 0.023 | 0.214 | Multiracial | Asian |
| 1022 | 0.503 | 0.002 | 0.496 | Multiracial | Asian |
| 1023 | 0.493 | 0.015 | 0.492 | Multiracial | Asian |
| 1024 | 0.501 | 0.494 | 0.005 | Other | Black |
| 1025 | 0.542 | 0.064 | 0.394 | Other | Black |
| "White American Indian" | | | | | |
| 1026 | 0.979 | 0.003 | 0.018 | White | American Indian |
| 1027 | 0.990 | 0.002 | 0.008 | White | American Indian |
| 1028 | 0.996 | 0.002 | 0.002 | White | American Indian |

[a] "Misreporting" in this context means a large gap exists between the respondent's self-reported race and bio-ancestry score.

Table 7 presents respondents whose bio-ancestry scores, self-reported race at Waves I and III, and interviewer-identified race at Wave III do not match up in Add Health. For these respondents, single-best race is the same as multiracial-allowed race so only multiracial-allowed race is presented. In Add Health, there are two types of differences. “Misreporting” is the first. Two respondents in the top panel are in this category. They have very high Asian ancestry scores, 0.988 and 0.953 respectively, and both their Wave I race and interviewer-recorded race are “Asian.” But they reported as “White” at Wave III. In the bottom panel is the second type. Respondents in this category are mixed race according to their bio-ancestry scores and reported different races at different waves.

Without bio-ancestry scores it is almost impossible to distinguish “misreporting” and mixed race in Tables 6 and 7, and data users would have to treat the two groups the same. In Table 6, for example, two respondents (Pseudo IDs 1001 and 1003) have the same pattern—online race is “White” and housing race is “Black”. It turns out that the respondent with Pseudo ID 1001 has a high African ancestry of 0.994, but the respondent with Pseudo ID 1003 possesses an African ancestry of 0.817 and a European ancestry of 0.170.

TABLE 1.7
All Respondents Who Differ among Bio-ancestry Score, Self-reported Race and Interviewer-reported Race, Add Health

| | Bio-ancestry Score | | | Self-reported Race | | Interviewer-reported Race |
|---|---|---|---|---|---|---|
| Pseudo ID | European | African | East Asian | Wave I | Wave III | Wave III |
| "Misreporting" [a] | | | | | | |
| 1001 | 0.008 | 0.004 | 0.988 | Asian | White | Asian |
| 1002 | 0.035 | 0.012 | 0.953 | Asian | White | Asian |
| Mixed Race | | | | | | |
| 1003 | 0.863 | 0.119 | 0.018 | White | Black | White |
| 1004 | 0.843 | 0.030 | 0.127 | White | Asian | American Indian |
| 1005 | 0.657 | 0.325 | 0.018 | Black | Black | Black |
| 1006 | 0.542 | 0.437 | 0.021 | Black | Black | Black |
| 1007 | 0.400 | 0.585 | 0.015 | Black | Asian | American Indian |
| 1008 | 0.420 | 0.053 | 0.527 | Asian | White | Asian |
| 1009 | 0.270 | 0.117 | 0.614 | American Indian | White | White |
| 1010 | 0.315 | 0.061 | 0.624 | American Indian | White | American Indian |
| 1011 | 0.341 | 0.004 | 0.654 | Other | White | White |
| 1012 | 0.475 | 0.010 | 0.515 | Other | White | White |
| 1013 | 0.386 | 0.070 | 0.544 | Other | White | White |
| 1014 | 0.384 | 0.120 | 0.496 | Other | White | White |
| 1015 | 0.358 | 0.202 | 0.440 | Other | White | White |
| 1016 | 0.347 | 0.173 | 0.481 | Other | White | White |
| 1017 | 0.249 | 0.389 | 0.361 | Other | White | White |
| 1018 | 0.023 | 0.341 | 0.636 | Other | Black | Black |
| 1019 | 0.828 | 0.153 | 0.020 | Other | Black | Black |

[a] "Misreporting" in this context means a large gap exists between the respondent's self-reported race and bio-ancestry score.

## The Sex Chromosomes and Self-reported Sex

The Sex Chromosomes and Sex

Genetic information on sex is not as certain as that for genetic relationships and bio-ancestry due to the complex human sex-determining process and our limited understanding of it (Ellegren 2011). XX and XY are the common combinations of sex chromosomes for females and males respectively in humans. The gonads need to be formed and differentiated for humans to develop sexually. The Y chromosome induces testis formation and is the dominant determinant for male development. Without a Y chromosome, gonads differentiate into ovaries, resulting in female development. On the human Y chromosome, the Sex-determining Region of the Y chromosome (SRY) plays the crucial role. The SRY is a single exon gene. In this gene there are transcription initiation sites and these transcripts are identified in adult testis and other male tissues (Clepet, Schater, Sinclair, Palmer, Lovell-Badge, and Goodfellow 1993). The SRY initiates testis development from early bipotential gonads (Koopman, Gubbay, Vivian, Goodfellow, and Lovell-Badge 1991; Sinclair, Berta, Palmer, Hawkins, Griffiths, Smith, Foster, Frischauf, Lovell-Badge, and Goodfellow 1990). The complex sex determination process also involves genes on autosomes. For example, *DMRT1*, which is located on chromosome 9, is found to be associated with haploinsufficiency in the form of XY sex reversal (Raymond, Parker, Kettlewell, Brown, Page, Kusz, Jaruzelska, Reinberg, Flejter, Bardwell, Hirsch, and Zarkower 1999).

Different combinations of the sex chromosomes other than XX and XY exist in the human population. XXY or XXYY males, known as Klinefelter syndrome, have two X chromosomes and one or two Y chromosomes. Also, persons with Turner syndrome have only one sex chromosome—that is, 45 chromosomes in total including only one X chromosome. The proportion of non-XX females and non-XY males is considerable. About 1 out of 500 to 1,000 boys are born with Klinefelter syndrome (Chen 2005), and 1 of every 2,700 live births result in a child with Turner syndrome (Jaffe 1999). Given the various forms of sex chromosome combinations, we maintain a cautious view on the application of genetic sex information in social surveys.

Due to the existence of different forms of sex chromosomes, it is impossible to be 100 percent certain of an individual's sex by only looking at the sex chromosomes. However, given the relatively low prevalence of unusual combination forms of sex chromosomes, the common forms of XX for females and XY for males may still serve as a reference.

Identifying "Misreported" Sex

In ROOM, 19 SNPs on X chromosomes and no SNPs on Y chromosomes were genotyped. So the genetic data is not entirely informative. X chromosomes are the only available information to suggest, rather than determine, biological sex.

One respondent who "misreported" sex is successfully identified using the sex chromosomal data in ROOM. The word "misreported" here refers to the situation in which self-reported sex differs from what biological sex and other information suggest. This respondent reported as male in the online survey but as female when applying for housing, and this person's roommate was female. It turns out 9 of 19 SNPs on the X chromosomes of this participant are heterozygous, which suggests a high probability of being female. Genetic sex, roommate sex, and housing application sex all indicate this is a female.

As mentioned previously, the sex chromosomal data also help identify misrepresented participant. In ROOM there is one pair of participants with identical genetic data, that is, with a proportion IBD score of 1. Information on the sex chromosomes suggests a high probability that the female in this pair gave saliva twice because 6 of 19 SNPs on the X chromosomes are heterozygous. The respondent's online survey sex and the roommate's sex are both female (roommate sex should be concordant with ego sex because of the housing policies), confirming that this participant is female.

**Discussion and Conclusion**

Using data from ROOM and Add Health, we show that proportion IBD score can identify “misreported” and unreported sibling type and detect misrepresented participants; bio-ancestry score can in some circumstances help to save missing race, and reconcile discrepancies among different measures of self-reported race; and the sex chromosomes offer information that, coupled with other variables, is helpful in checking gender. In some cases, only with the genetic information can the issues be successfully detected and recovered. An example is that Table 6 shows that without the bio-ancestry scores it is almost impossible to distinguish between respondents who “misreported” race and those who are mixed-race.

The cost of using already-collected DNA data to implement quality control is minimal, but the benefits of doing so are considerable. A total number of 127 cases with various data issues can be repaired and recovered with the help of the genetic information: Six pairs of “misreported” MZ twins are found; five twin pairs of uncertain status are determined; one misrepresented respondent is corrected; 59 missing values in the race variables can be saved; four respondents who “misreported” their race are found; 40 discrepancies in the race variables are clarified; and one respondent who “misreported” sex is found. Particularly, for studies that analyze roommate pairs or sibling pairs, recovering one respondent means a pair can be saved. Moreover, tremendous amounts of financial support, time, personnel and other resources are invested in social surveys. If a large-scale survey spends $100 million to collect data on 20,000 individuals, this represents an average of $5,000 per participant. In this sense, it is worthwhile to repair every data issue possible.

We emphasize that genetic data are not a substitute for self-reported data. Sibling type, race and gender are all socially and biologically constructed in a complex way. The fact that respondents in ROOM and Add Health, most of whom are adolescents and young adults, change or do not report these variables at different waves should be understood from a sociological perspective in combination with other information. Social scientists have long been interested in racial and ethnic identities among adolescents and adults (e.g., Phinney 1990). Identity development (Erikson 1968), social identity theory (Tajfel and Turner 1979) and acculturation theory (Park 1928; Stonequist 1937) provide theoretical frameworks for understanding why people may change racial and ethnic identities (for a review see Frable 1997). From the social constructionist perspective gender identity is conceptualized as an ongoing performance. Individuals enact and reinforce their femininity/masculinity in social interactions (West and Zimmerman 1987). Therefore, when a respondent “misreports” her race or gender on a questionnaire, it is possible that the respondent has changed her racial or gender identity. As shown in this article, with DNA data we can obtain more sociologically interesting information concerning race and gender.

Genetic information may reveal personal privacy or be used to trace or identify participants (Gymrek, McGuire, Golan, Halperin, and Erlich 2013). Careful practice should be made when working with genetic data. National Research Council (Council 2010) provides recommendations regarding new issues raised by collecting biological data including protecting privacy, obtaining consent, and sharing data among other important issues. Researcher should be fully prepared for the issues and potential challenges in order to properly use DNA data in social surveys.

The relatively small numbers of SNPs genotyped in ROOM (350) and Add Health (1,140) limit the findings of this article in several ways. First, IBD score is traditionally estimated from pedigrees and the base population, which is the population that consists of the founders of the pedigrees. Ideally, with large SNP datasets IBD can be predicted without knowing pedigrees. However, because of the relatively small numbers of SNPs, the IBD score estimated here may not be as accurate as that obtained from dense SNP data. For the same reason, an alternative calculation of IBD score is used in this article. It should be noted that calculating IBD score can be more complicated than the calculation in this article (see Powell, Visscher, and Goddard 2010 for a review on IBD). Second, the AIMs selected in this research detect three major continental populations; however we were not able to break down the African, East Asian and European classifications into more specific groups. Third, as for the sex chromosomes, only X chromosomes were genotyped and the numbers of genotyped SNPs on the X chromosomes are small. Given the complex nature of sex determination process, information about Y chromosomes and more SNPs on X chromosomes are desirable in the future.

This article provides an initial effort to improve survey data using genetic data. Rapid developments in genetics and the increasing collection of genetic data will introduce more ways to improve data quality. Recently, large-scale social surveys such as the Health and Retirement Study (Crimmins et al. 2009) started to release genotype data that contain millions of SNPs. The large datasets provide opportunities for further exploration of innovative methods for data quality control.

REFERENCES


Caspi, Avshalom, Joseph McClay, Terrie E. Moffitt, Jonathan Mill, Judy Martin, Ian W. Craig, Alan Taylor, and Richie Poulton. 2002. "Role of Genotype in the Cycle of Violence in Maltreated Children." *Science* 297:851-854.

Chen, Harold. 2005. "Klinefelter Syndrome." vol. 2014, edited by B. Buehler.

Clepet, Christian, Alan J. Schater, Andrew H. Sinclair, Mark S. Palmer, Robin Lovell-Badge, and Peter N. Goodfellow. 1993. "The human SRY transcript." *Human Molecular Genetics* 2:2007-2012.

Council, National Research. 2010. *Conducting Biosocial Surveys: Collecting, Storing, Accessing, and Protecting Biospecimens and Biodata*. Washington, D.C.: The National Academies Press.

Crimmins, Ellen M., Heidi Guyer, Kenneth M. Langa, Mary Beth Ofstedal, Robert B. Wallace, and David R. Weir. 2009. "Documentation of Biomarkers in the Health and Retirement Study." vol. 2014.

Domingue, BW, J Fletcher, D Conley, and JD Boardman. 2014. "Genetic and educational assortative mating among US adults." *Proceedings of the National Academy of Sciences* 111:7996-8000.

Ellegren, Hans. 2011. "Sex-chromosome evolution: recent progress and the influence of male and female heterogamety." *Nature Reviews Genetics* 12:157-166.

Enoch, M., P. Shen, K. Xu, C. Hodgkinson, and D. Goldman. 2006. "Using ancestry-informative markers to define populations and detect population stratification." *Journal of Psychopharmacology* 20:19-26.

Erikson, E. H. 1968. *Identity: Youth and Crisis*. New York: Norton.

Eschbach, Karl. 1993. "Changing Identification among American Indians and Alaska Natives." *Demography* 30:635-652.

Frable, Deborrah E. S. 1997. "Gender, Racial, Ethnic, Sexual, and Class Identities." *Annual Review of Psychology* 48:139-162.

Friedlaender, J. S., F. R. Friedlaender, F. A. Reed, K. K. Kidd, J. R. Kidd, G. K. Chambers, R. A. Lea, J. H. Loo, G. Koki, J. A. Hodgson, D. A. Merriwether, and J. L. Weber. 2008. "The genetic structure of Pacific islanders." *Plos Genetics* 4:173-190.

Frudakis, T., K. Venkateswarlu, M. J. Thomas, Z. Gaskin, S. Ginjupalli, S. Gunturi, V. Ponnuswamy, S. Natarajan, and P. K. Nachimuthu. 2003. "A classifier for the SNP-Based inference of ancestry." *Journal of Forensic Sciences* 48:771-782.

Guo, Guang, Jessica Halliday Hardie, Craig Owen, Jonathan K. Daw, Yilan Fu, Hedwig Lee, Amy Lucas, Emily McKendry-Smith, and Greg Duncan. 2009. "DNA Collection in a

Social Science Study: A Pilot Study of Peer Impacts on Attitudes and Drinking Behavior." *Sociological Methodology* 39:1-29.

Gymrek, Melissa, Amy L. McGuire, David Golan, Eran Halperin, and Yaniv Erlich. 2013. "Identifying Personal Genomes by Surname Inference." *Science* 339:321-324.

Halder, I., M. Shriver, M. Thomas, J. R. Fernandez, and T. Frudakis. 2008. "A panel of ancestry informative markers for estimating individual biogeographical ancestry and admixture from four continents: Utility and applications." *Human Mutation* 29:648-658.

Harris, Kathleen Mullan. 2013. "Design Features of Add Health." vol. 2014.

Hernandez, Lyla M. and Dan G. Blazer. 2006. *Genes, Behavior, and the Social Environment: Moving Beyond the Nature/Nurture*. Washington, DC: National Academies Press.

Hitlin, Steven, J. Scott Brown, and Glen H. Elder. 2006. "Racial self-categorization in adolescence: Multiracial development and social pathways." *Child Development* 77:1298-1308.

Jaffe, Robert B. 1999. "Disorders of Sexual Development." in *Reproductive endocrinology: physiology, pathophysiology, and clinical management*, edited by S. S. C. Yen, R. B. Jaffe, and R. L. Barbieri: Philadephia: W.B. Saunders.

Kelly, Mary E. and Joane Nagel. 2002. "Ethnic re-identification: Lithuanian Americans and Native Americans." *Journal of Ethnic and Migration Studies* 28:275-289.

Koopman, Peter, John Gubbay, Nigel Vivian, Peter Goodfellow, and Robin Lovell-Badge. 1991. "Male Development of Chromosomally Female Mice Transgenic for Sry." *Nature* 351:117-121.

Malécot, G. 1948. *Les Mathématiques de L'Hérédité*. Paris: Masson & Cie.

Mitchell, Colter, Daniel Notterman, Jeanne Brooks-Gunn, John Hobcraft, Irwin Garfinkel, Kate Jaeger, Iulia Kotenko, and Sara McLanahan. 2011. "Role of mother's genes and environment in postpartum depression." *Proceedings of the National Academy of Sciences* 108:8189-8193.

Park, R. 1928. "Human Migration and the Marginal Man." *American Journal of Sociology* 33:881-893.

Parra, E. J., A. Marcini, L. Akey, J. Martinson, M. A. Batzer, R. Cooper, T. Forrester, D. B. Allison, R. Deka, R. E. Ferrell, and M. D. Shriver. 1998. "Estimating African American admixture proportions by use of population-specific alleles." *American Journal of Human Genetics* 63:1839-1851.

Phinney, Jean S. 1990. "Ethnic Identity in Adolescents and Adults: Review of Research." *Psychological Bulletin* 108:499-514.

Powell, Joseph E., Peter M. Visscher, and Michael E. Goddard. 2010. "Reconciling the analysis of IBD and IBS in complex traits studies." *Nature Reviews Genetics* 11:800-805.

Pritchard, J. K., M. Stephens, and P. Donnelly. 2000. "Inference of population structure using multilocus genotype data." *Genetics* 155:945-959.

Purcell, Shaun, Benjamin Neale, Kathe Todd-Brown, Lori Thomas, Manuel A.R. Ferreira, a David Benderb, Julian Maller, Pamela Sklar, Paul I.W. de Bakker, Mark J. Daly, and Pak C. Sham. 2007. "PLINK: A Tool Set for Whole-Genome Association and Population-Based Linkage Analyses." *American Journal of Human Genetics* 81:559-575.

Raymond, Christopher S., Emily D. Parker, Jae R. Kettlewell, Laura G. Brown, David C. Page, Kamila Kusz, Jadwiga Jaruzelska, Yuri Reinberg, Wendy L. Flejter, Vivian J. Bardwell, Betsy Hirsch, and David Zarkower. 1999. "A region of human chromosome 9p required for testis development contains two genes related to known sexual regulators." *Human Molecular Genetics* 8:989-996.

Reichman, Nancy E., Julien O. Teitler, Irwin Garfinkel, and Sara S. McLanahan. 2001. "Fragile Families: sample and design." *Children and Youth Services Review* 23:303-326.

Rosenberg, N. A., J. K. Pritchard, J. L. Weber, H. M. Cann, K. K. Kidd, L. A. Zhivotovsky, and M. W. Feldman. 2002. "Genetic structure of human populations." *Science* 298:2381-2385.

Shanahan, Michael, Stephen Vaisey, Lance D. Erickson, and Andrew Smolen. 2008. "Environmental Contingencies and Genetic Propensities: Social Capital, Educational Continuation, and Dopamine Receptor Gene DRD2." *American Journal of Sociology* 114:s260-s286.

Shriver, M. D., M. W. Smith, L. Jin, A. Marcini, J. M. Akey, R. Deka, and R. E. Ferrell. 1997. "Ethnic-affiliation estimation by use of population-specific DNA markers." *American Journal of Human Genetics* 60:957-964.

Sinclair, Andrew H., Philippe Berta, Mark S. Palmer, J. Ross Hawkins, Beatrice L. Griffiths, Matthijs J. Smith, Jamie W. Foster, Anna-Maria Frischauf, Robin Lovell-Badge, and Peter N. Goodfellow. 1990. "A gene from the human sex-determining region encodes a protein with homology to a conserved DNA-binding motif." *Nature* 346:240-244.

Smith, M. W., J. A. Lautenberger, H. D. Shin, J. P. Chretien, S. Shrestha, D. A. Gilbert, and S. J. O'Brien. 2001. "Markers for mapping by admixture linkage disequilibrium in African American and Hispanic populations." *American Journal of Human Genetics* 69:1080-1094.

Stonequist, E. 1937. *The Marginal Man: A Study in Personality and Culture Conflict*. New York: Scribner's Sons.

Tajfel, H. and J. Turner. 1979. "An integrative theory of intergroup conflict." in *The Social Psychology of Intergroup Relations*, edited by W. Austin and S. Worchel. Monterey, CA: Brooks/Cole.

Wang, S., C. M. Lewis, M. Jakobsson, S. Ramachandran, N. Ray, G. Bedoya, W. Rojas, M. V. Parra, J. A. Molina, C. Gallo, G. Mazzotti, G. Poletti, K. Hill, A. M. Hurtado, D. Labuda, W. Klitz, R. Barrantes, M. C. Bortolini, F. M. Salzano, M. L. Petzl-Erler, L. T. Tsuneto, E. Llop, F. Rothhammer, L. Excoffier, M. W. Feldman, N. A. Rosenberg, and A. Ruiz-Linare. 2007. "Genetic variation and population structure in Native Americans." *Plos Genetics* 3:2049-2067.

West, C. and D. Zimmerman. 1987. "Doing Gender." *Gender and Society* 1:125-151.

Wright, Sewall. 1917. "Coefficients of inbreeding and relationship." *American Naturalist* 56:330-338.

Yang, N., H. Z. Li, L. A. Criswell, P. K. Gregersen, M. E. Alarcon-Riquelme, R. Kittles, R. Shigeta, G. Silva, P. I. Patel, J. W. Belmont, and M. F. Seldin. 2005. "Examination of ancestry and ethnic affiliation using highly informative diallelic DNA markers: application to diverse and admixed populations and implications for clinical epidemiology and forensic medicine." *Human Genetics* 118:382-392.

# CHAPTER 2 –DOES MARRIAGE MODERATE GENETIC EFFECTS ON DELINQUENCY AND VIOLENCE?

## Introduction

The impact of marriage on individuals' well-being has long been studied. Married individuals exhibit higher levels of healthy behaviors, survival probability, wages, and so forth than unmarried individuals (Waite 1995). Of particular interest has been the inhibiting effect of marriage on antisocial behavior such as delinquency and crime. Studies have found that the transition to marriage is linked to a decline in antisocial behavior. This desistance effect of marriage is noted in multiple cohorts (King, Massoglia, and Macmillan 2007; Sampson and Laub 1993), and in different countries (Blokland and Nieuwbeerta 2005; Theobald and Farrington 2009).

In recent years, researchers have increasingly incorporated genetic variables to examine the effects of social environments on antisocial behavior (e.g., Caspi, McClay, Moffitt, Mill, Martin, Craig, Taylor, and Poulton 2002). The findings that social factors interact with genes to influence antisocial behavior underline the importance of gene-environment interaction (G×E) (G×E refers to processes wherein genetic influences depend on environmental factors, or vice versa). But existing G×E research almost exclusively focuses on one or a few genetic variants. Unlike rare Mendelian traits that are determined by a single gene or allele, overall genetic influence on antisocial behavior comprises a large number of genetic effects (Anholt and Mackay 2012). Therefore, it is essential to examine more than a few genetic variants in G×E research on antisocial behavior.

We extended previous G×E research by considering a large number of genetic variants.

Drawing on data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), we examined whether marriage moderates the collective effect of 580 single nucleotide polymorphisms (SNPs) in 64 genes on delinquency and violence. To do so we employed a recently developed mixed linear model implemented in the genome-wide complex traits analysis (GCTA) software (Yang, Lee, Goddard, and Visscher 2011). This new method estimates a heritability parameter—the proportion of variance in the phenotype that is jointly explained by the SNPs. We examined the gene-by-marriage interaction by comparing the proportion of variance in antisocial behavior explained by 580 SNPs among married and unmarried individuals. The samples in the mixed linear models were drawn from the Add Health genetic subsample. Our approach could be implemented using larger data in the future. Selection, confounding and heterogeneity can bias the estimate of the gene-by-marriage interaction. We conducted a series of analyses to address these issues.

## Background

### Marriage and Antisocial Behavior

Social scientists have long noticed that marriage is an important life-course transition with seemingly far reaching impact. In general, married individuals consider marriage a long-term contract (Waite 1995). To maintain the contract married individuals tend to do things that pay off in the long run, and refrain from behaviors that bring instant gratifications or the possibility of harmful consequences. This is supported by the findings that marriage may deter criminal activity and deviant behavior (e.g., Blokland and Nieuwbeerta 2005; Farrington and West 1995; Horney, Osgood, and Marshall 1995; Sampson and Laub 1993; Warr 1998).

The effect of marriage on antisocial behavior may be thought of as the result of three processes. First, marriage may strengthen connections within the family. Married couples are connected to each other in relationships for which there are strong social norms. Married people tend to fulfill normative expectations implied by marriage. Derived from social control theory (Hirschi 1969), Sampson, Laub and colleagues focused on bonds and ties created within marriage (Laub, Nagin, and Sampson 1998; Laub and Sampson 2003; Sampson and Laub 1993; Sampson, Laub, and Wimer 2006). In this line of research, it is proposed that marriage establishes strong bonds and ties that prevent individuals from committing crime over the life course. Interpersonal attachment to a partner serves as a control mechanism. Over time individuals invest more and more socially and financially in a marriage. Engaging in criminal activity is not a rational choice because it threatens that investment. Summarizing the position, Sampson and Laub (1993: 141) stated that marriage creates "interdependent systems of obligation and constraints that impose significant costs for translating criminal propensities into action."

Second, marriage may weaken connections outside of the family that might lead to antisocial behavior. Peer influence can be a major source of variation in antisocial behavior (Osgood, Wilson, O'Malley, Bachman, and Johnston 1996). The transition to marriage usually means that routine activities are primarily devoted to the spouse and family. Warr (1998) showed that marriage may weaken or disrupt connections with peers including delinquent ones. Following the transition to marriage, time spent with peers decreases dramatically. As a result, opportunities and motivations to engage in crime and delinquency are significantly limited. Warr found that these changes largely account for the association between marriage and antisocial behavior. In addition, obligations that come with marriage tend to leave less time for leisure activities outside of the family (Osgood and Lee 1993). As such, unstructured socializing with delinquent peers may also be limited.

Third, marriage may lead to changes at the psychological level and, by extension, alter one's perception of antisocial behavior. Because marriage implies meaningful commitment, married persons may develop a sense of obligation to their partners that reduces the appeal of behaviors that might threaten the relationship. Cognitive and identity transformations are at work when individuals desist from antisocial behavior (Giordano, Cernkovich, and Rudolph 2002). After getting married, individuals are open to make cognitive changes and treat the relationship seriously. For example, stealing and drug use are no longer viewed proper and viable. Consequently, deviant behavior is less likely to occur. Emotional regulation is also important to the success of desistance (Giordano, Schroeder, and Cernkovich 2007). Negative emotions associated with crime, coupled with the ability to manage emotions, may lead to a decline in criminal activity. An implication of these findings is that marriage might involve changes in emotional regulation that help individuals desist.

Gene-Environment Interaction Research on Antisocial Behavior

G ×E studies on antisocial behavior have focused on five genes—the monoamine oxidase A (MAOA) gene, the dopamine D2 receptor (DRD2) gene, the serotonin transporter gene (5-HTT), the dopamine receptor gene (DRD4), and the dopamine transporter gene (DAT1). Using data from the Dunedin Multidisciplinary Health and Development study, Caspi and colleagues (2002) reported that the effect of childhood maltreatment on antisocial behavior is weaker among individuals with high MAOA activity than those with low MAOA activity. Using data from Add Health, Guo and colleagues (2008) found that the effects of the DRD2 and MAOA genes on delinquency are conditional on family processes, school processes and social networks. Recently, Simons and colleagues (2011) found that the presence of both short allele in the 5-HTT gene and long allele in the DRD4 gene interacts with social environments to affect aggression.

Marriage is an important social institution that may also moderate genetic effects on antisocial behavior. To date only one study examined the gene-by-marriage interaction on delinquency (Beaver, Wright, DeLisi, and Vaughn 2008). The authors tested the interactions between marriage and five genes, the DAT1, DRD2, DRD4, 5-HHT, and MAOA genes, using the Add Health data. The authors found significant interactions (at the 0.10 level) only among males. The temporal order between marriage and delinquency was not considered.

Genetic Effects on Antisocial Behavior

In the aforementioned G×E studies, genetic effects are represented by only a few genetic variants. Antisocial behavior, however, is influenced by a large number of genes (Craig and Halton 2009). Researchers have identified numerous genes and biological mechanisms related to antisocial behavior in the human population. Genetic analyses have implicated the MAOA (Manucka, Flory, Ferrell, Mann, and Muldoon 2000), SLC6A4 (Murphy, Fox, Timpano, Moya, Ren-Patterson, Andrews, Holmes, Lesch, and Wendland 2008), TPH1 (Hennig, Reuter, Netter, Burk, and Landt 2005), 5-HT1B hetero-receptors (Soyka, Preuss, Koller, Zill, and Bondy 2004), Dopamine-β-hydroxylase (DβH) (Hess, Reif, Strobel, Boreatti-Hümmer, Heine, Lesch, and Jacob 2009), and GABA neurotransmitters (Miczek, Fish, Bold, and Almeida 2002) among many others in predisposition towards aggression, delinquency, and violent behavior in human populations (for a review see Craig and Halton 2009). Possible biological mechanisms include cortisol levels that monitor the hypothalamus, pituitary and adrenal (HPA) axis (Shirtcliff, Granger, Booth, and Johnson 2005), levels of the serotonin metabolite 5-hydroxy-indole acetic acid (5-HIAA) in cerebrospinal fluid (CSF) (Coccaro, Kavoussi, Trestman, Gabriel, Cooper, and Siever 1997), and potentially serotonin mechanisms, insulin levels and glucose metabolism

(Linnoila and Virkkunen 1992).

Studying model organisms can help identify genes for antisocial behavior in humans. Humans and nonhuman animals share neurochemical and anatomical systems that are activated when aggressive behavior occurs (Nelson and Trainor 2007). Rodents are among the ideal animals that can be studied to provide new knowledge for genetics of aggression. About 90% of genes in rats are orthologous to genes in humans (Consortium 2004). In addition, the phenotype of rodents can be measured more precisely, and the genetic background and environmental conditions can be controlled more easily. Anholt and Mackay (2012) reported that researchers successfully identify genes and pathways that influence aggression by employing quantitative trait locus mapping and analysis of single-gene mutations in mice. In our analysis, 39 genes are known to be related to aggression in mice.

Selection, Confounding, and Population Heterogeneity

Marriage is not a random event. Issues such as selection, confounding, and population heterogeneity may pose threats to the marriage-antisocial-behavior association, thereby undermining the validity of the gene-by-marriage interaction results. Differential selection is one of the largest threats to claim a causal effect of marriage (e.g., King, Massoglia, and Macmillan 2007; Sampson, Laub, and Wimer 2006). Suppose that delinquent persons self-select out of marriage—either by remaining single or being more likely to divorce. Then it is not marriage that makes individuals less antisocial, and the observation that genetic effects for delinquency depend on marital status possibly just reflects the difference in genetic effects between delinquent and nondelinquent persons.

Age may have a confounding effect on the inhibiting effect of marriage. Delinquency usually peaks during adolescence and young adulthood, and declines dramatically thereafter

(Hirschi and Gottfredson 1983). In other words, along with a decline in antisocial behavior most people experience major changes in life circumstances such as marriage. Thus, it can simply be that older individuals are more likely to get married and less likely to act antisocially. In this scenario, the interaction effect of marriage could merely represent the effect of age or maturity.

A third issue involved in the desistance process is that the effect of marriage may not be universal for every individual due to population heterogeneity, which refers to the situation in which individuals differ in propensity to commit deviant behavior (DeLisi 2005; Nagin and Paternoster 2000). Moffitt (1993) argued that in a population one group of individuals repeatedly engages in deviant behavior over the life course (persistent offenders), whereas the remaining individuals act delinquently primarily during adolescence. Persistent offenders do not practice much prosocial behavior during early childhood. As a result, marriage may not have as much impact on persistent offenders as it does on others. The gene-by-marriage interaction, therefore, may vary in magnitude for persistent and nonpersistent offenders. In this article, we conducted analyses to examine whether the effect of marriage is threatened by the three issues.

### Research Question and Hypothesis

As discussed above, marriage may foster desistance by strengthening bonds within the family, weakening antisocial ties outside of the family, and altering one's psychological perception of deviant behavior. Taking genetic influences into account, this article further explored the role of marriage in the desistance process. We examined whether marriage can inhibit delinquency and violence through a biological pathway—the modification of a large number of genetic effects for antisocial behavior. Given that existing literature suggests that marriage has an inhibiting effect on antisocial behavior, we hypothesized that the collective influence of the genes on antisocial behavior was smaller among married individuals than that of unmarried individuals.

**Data, Measures and Method**

Data

Our analysis used the genetic subsample of Add Health. Add Health is a nationally representative sample of U.S. adolescents in grades 7-12 in 1994-95 (Harris, Halpern, Whitsel, Hussey, Tabor, Entzel, and Udry 2009). The first wave of data collection took place in the 1994-95 school year. A sample of about 20,000 adolescents was drawn. Respondents were surveyed through in-school questionnaires and in-home interviews. Three subsequent waves of data were collected at respondents' homes in 1996 (Wave II), 2001-02 (Wave III), and 2008-09 (Wave IV). A wide range of information including social background and behaviors was collected at each wave. In addition, the data has rich information on parents and romantic partners.

The genetic subsample consisted of 2,612 respondents identified as siblings or twins at Wave I. At Wave III saliva of the genetic subsample was collected and genotyped. DNA was isolated from buccal cells at the Institute of Behavior Genetics at the University of Colorado, Boulder. The average yield of DNA was 58 $\pm 1$ µg. The genotype data were based on an Illumina GoldenGate assay. The GoldenGate array targeted 1,536 SNPs. A total of 1,140 SNPs in 130 genes were successfully genotyped. The number of respondents whose DNA was successfully genotyped was 2,281. The 2,281 respondents came from 1,428 families. Of the 1,428 families, 770 included two children both of whom had genotype data, 33 included three children all of whom had genotype data, and two families included four children all of whom have genotype data. There were 623 families in which only one child had genotype data, although this child had sibling(s) or a twin. We selected 580 SNPs in 64 autosomal genes for the current analysis. Of the

64 genes, 39 genes reviewed and summarized by Maxson (2009) are associated with aggression in transgenic or knock-out studies of mice, and 25 genes are related to risky behavior such as drinking and drug use in the human population.

Delinquency and Violence

A 4-item nonviolence scale and an 8-item violence scale were used to measure delinquency and violence respectively. The nonviolence delinquency included stealing amounts larger or smaller than $50, breaking and entering, and selling drugs within the past 12 months. Violence included serious physical fighting result in the need for medical treatment, use of weapons to get something from someone, physical fighting between groups, shooting or stabbing someone, deliberately damaging property, carrying a weapon (unavailable at Wave IV), and pulling a knife or gun on someone within the past 12 months. The sum of delinquency and violence was treated as the third dependent variable. The two scales are a variation of a scale that is widely used in research on delinquency and crime (Thornberry and Krohn 2000).

Desistance

Desistance can be defined either as a process or an end state (Laub and Sampson 2001). Treating desistance as a process requires more frequent assessments of the behavior, whereas treating desistance as an end state requires a longer time frame (Mulvey, Steinberg, Fagan, Cauffman, Piquero, Chassin, Knight, Brame, Schubert, Hecker, and Losoya 2004). Similar to the study by Glueck and Glueck (1950) that interviewed subjects at an average age of 14, 25, and 32, Add Health collected information from participants at an average age of 15, 22, and 28. Given the relatively frequent assessments from adolescence to young adulthood among Add Health participants, we were able to study desistance as a process.

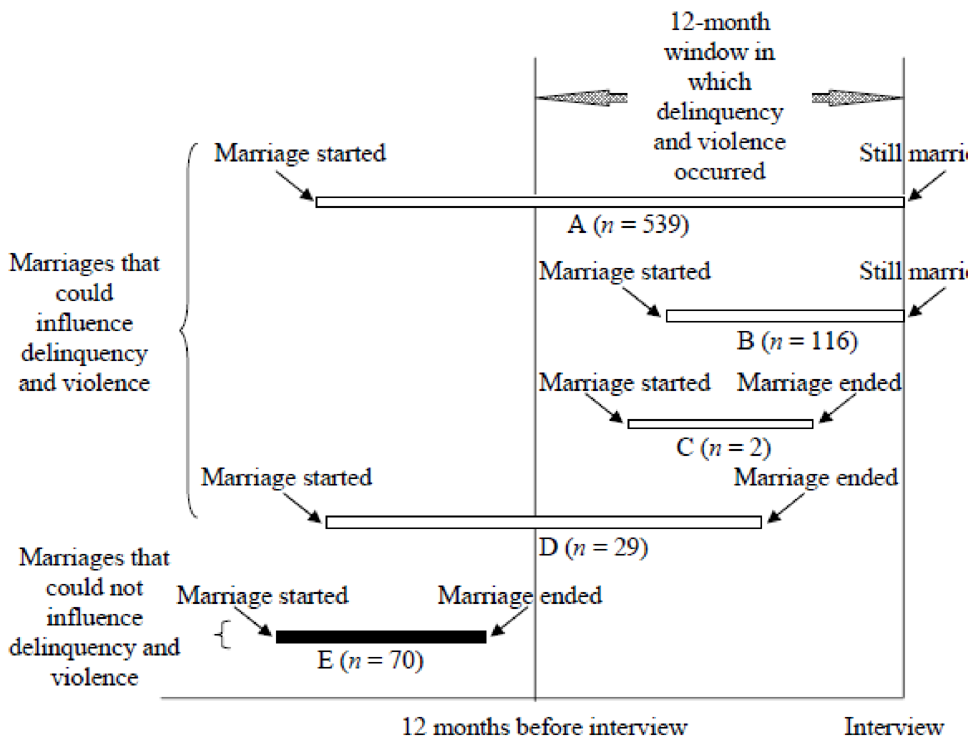
Following the majority of research (e.g., Horney, Osgood, and Marshall 1995; Laub, Nagin, and Sampson 1998; Theobald and Farrington 2009; Warr 1998), we assumed that only individuals who were delinquent in the first place were eligible for desisting from delinquency and violence. Respondents who scored at least 1 on either the delinquency or violence scale at Waves I and II were included in our sample. The final sample consisted of 1,254 individuals.

Reports from Bureau of Justice Statistics (2001-09) showed that the percentage of the U.S. adult population under age 35 that was or had ever been incarcerated in a state or federal prison or in a local jail ranged from about 1% to 4%, and this percentage was remarkably higher among the 20 and older age group than the 18-19 age group. As a result, chronic offenders, especially those who were 20 and older, may be more likely to drop out of the study. In Add Health, due to incarceration about 12 individuals from the genetic sample were not interviewed at Wave III. Therefore, conclusions based on the sample may not necessarily apply to the correctional population.

Marriage and Its Temporal Relation with Delinquency and Violence

To isolate the effect of marriage, it is crucial to sort out the temporal order between marriage and delinquency and violence. At Waves III and IV, respondents were asked to report the number of times they had been married and the start and end dates of each marriage. But we only knew of delinquent and violent behaviors that occurred in the 12 months before the interview. No exact timing of the behaviors within this 12-month window was available. Figure 1 is an illustration of how marital status was defined. We divided marriages into two groups based on whether the marriage ended before the 12-month window. The first group of marriages (types A to D represented by the white lines) overlapped the 12-month window. We assumed that these marriages could influence delinquency and violence that occurred during this timeframe. The other group of marriages (type E represented by the black line) were those that ended at least 12 months prior to the interview. We assumed that these marriages could not influence delinquency and violence during this timeframe.

Figure 2.1
Temporal order between marriage and delinquency/violence: Distinguishing marriages that could influence delinquency/violence, and marriages that could not




represents marriages that overlapped the 12-month window, and could influence delinquency and violence that occurred in the 12-month window (types A through D).

represents marriages that ended 12 months before the interview, and could not influence delinquency and violence that occurred in the 12-month window (type E).

Note: *n* in parentheses indicates the number of marriages for each type. Two marriages were intact when interviewed but their start dates were missing. We considered these two marriages could influence delinquency and violence.

Most of the studies that reported the inhibiting effect of marriage used data in which respondents married in the 1950s, 1960s or 1980s (e.g., Farrington and West 1995; King, Massoglia, and Macmillan 2007; Laub and Sampson 2003). An advantage of the Add Health data is that we could test whether the marriage effect extended to a more recent cohort.

Marriage and Cohabitation

Mechanisms for antisocial behavior may be different between cohabitors and married persons (Horney, Osgood, and Marshall 1995). Therefore we first compared the levels of delinquent and violent behaviors in married, cohabitating, and single individuals. The results (not shown) suggested that cohabitors and single persons tended to report higher levels of antisocial behavior than married persons. Thus married individuals were coded as 1 and unmarried individuals, namely, cohabitating and single persons, were coded as 0.

Control Variables

Control variables included age, gender, race, education, employment, church going, household size, verbal IQ (PVT) score, parental education, closeness to parents, and bio-ancestry scores. Parental education was a family-level variable and the remaining controls were individual-level variables. Missing values in the control variables were imputed by the multiple imputation technique (Rubin 1987). The missing values were imputed five times to generate five complete data sets and then the regression results using the five complete data sets were combined to produce inferential results. We did not impute missing values in delinquency, violence, or marriage. The estimation of bio-ancestry scores (Pritchard, Stephens, and Donnelly 2000) relied on 121 ancestral informative markers that were used to distinguish three major continental populations—African, East Asian, and European. Each respondent was assigned three scores—African, East Asian, and European. The sum of the three scores was 1. Because using bio-ancestry scores to adjust for population stratification is a recommended method in genetic analysis (McCarthy, Abecasis, Cardon, Goldstein, Little, Ioannidis, and Hirschhorn 2008), we controlled for bio-ancestry scores in the mixed linear models. Replacing bio-ancestry scores with self-reported race yielded similar results as bio-ancestry scores were highly correlated with self-reported race (e.g., the average European bio-ancestry score for White was 0.95). Table 1 presents descriptive statistics and brief descriptions for the variables.

Table 2.1
Descriptive Statistics for All Variables

| | Wave I 1994-95 *N*=1,253-1,254 | Wave II 1996 *N*=1,196-1,254 | Wave III 2001-02 *N*=1,249-1,254 | Wave IV 2008-09 *N*=1,117-1,254 |
|---|---|---|---|---|
| | Mean | Mean | Mean | Mean |
| Delinquency | 1.05 | 0.80 | 0.44 | 0.21 |
| | (1.82) | (1.62) | (1.21) | (0.86) |
| Violence | 1.91 | 1.20 | 0.55 | 0.47 |
| | (2.65) | (2.05) | (1.31) | (1.08) |
| Marital status | | | | |
| Married | ---- | ---- | .16 | .43 |
| Unmarried | ---- | ---- | .84 | .57 |
| Age | 15.46 | 16.39 | 21.80 | 28.26 |
| | (1.60) | (1.62) | (1.64) | (1.68) |
| Gender[a] | | | | |
| Female | .42 | ---- | ---- | ---- |
| Male | .58 | ---- | ---- | ---- |
| Race[a] | | | | |
| American Indian | .03 | ---- | ---- | ---- |
| Asian | .06 | ---- | ---- | ---- |
| Black | .17 | ---- | ---- | ---- |
| Multiracial | .05 | ---- | ---- | ---- |
| Other | .01 | ---- | ---- | ---- |
| White | .67 | ---- | ---- | ---- |
| Bio-ancestry score[a] | | | | |
| African | 0.19 | ---- | ---- | ---- |
| | (.35) | | | |
| East Asian | 0.12 | ---- | ---- | ---- |
| | (.25) | | | |
| European | 0.70 | ---- | ---- | ---- |
| | (.39) | | | |
| Education | | | | |
| No college | ---- | ---- | .51 | .34 |
| College | ---- | ---- | .48 | .55 |
| Missing | ---- | ---- | .01 | .10 |
| Employment | | | | |
| Unemployed | ---- | ---- | .30 | .17 |
| Employed | ---- | ---- | .70 | .72 |
| Missing | ---- | ---- | .00 | .11 |
| Church going | | | | |

| | | | | |
|---|---|---|---|---|
| Less than weekly | .61 | .61 | .83 | .76 |
| Weekly or more | .38 | .33 | .16 | .13 |
| Missing | .02 | .06 | .01 | .10 |
| Household size | | | | |
| <3 | .01 | .08 | .29 | .30 |
| 3-6 | .67 | .66 | .58 | .51 |
| >6 | .33 | .26 | .13 | .08 |
| Missing | .00 | .00 | .00 | .11 |
| Verbal IQ (PVT) score | | | | |
| <90 | .23 | ---- | ---- | ---- |
| 90-110 | .48 | ---- | ---- | ---- |
| >110 | .26 | ---- | ---- | ---- |
| Missing | .03 | ---- | ---- | ---- |
| Parental education | | | | |
| Below high school | .12 | ---- | ---- | ---- |
| High school | .29 | ---- | ---- | ---- |
| More than high school | .55 | ---- | ---- | ---- |
| Missing | .04 | ---- | ---- | ---- |
| Closeness to parents[c] | | | | |
| Not Close | .39 | ---- | ---- | ---- |
| Close | .60 | ---- | ---- | ---- |
| Missing | .02 | ---- | ---- | ---- |

*Note*: Numbers in parentheses are standard deviations.

[a]The distributions of gender across four waves were almost identical. So were race and bio-ancestry score. Information at Wave I is presented for the three variables. [b]We imputed control variables to the maximum sample size—1,254. We did not impute delinquency, violence and marriage. [c]Not Close was defined as "somewhat", "very little" and "not at all" close to parents and close was defined as "very much" and "quite a bit" close to parents.

Modeling the Gene-by-Marriage Interaction

To model the interaction between 580 SNPs and marriage, we extended the mixed linear model implemented in the GCTA software (Yang, Lee, Goddard, and Visscher 2011). This model estimates the proportion of phenotypic variance that is accounted for by the linear, additive effects of the SNPs. Equation (2) below describes the basic structure of the mixed linear model.

$$Y=X\beta + W\mu + \varepsilon \qquad (2)$$

where Y is delinquency or violence; β is a vector of fixed effects for the control variables; μ is a vector of SNP effects with μi~ N (0, σ2μ) where i=1,…, N with N being the number of SNPs; ε is a vector of residual effects with $\varepsilon j \sim N(0, \sigma 2\varepsilon)$ where $j=1,\ldots,n$, with n being the number of individuals in the sample; and W is a standardized genotype matrix with the ijth element $wij = (sij- 2pi)/sqt[(2pi(1 - pi)]$ where sij is the number of copies of the reference allele for the ith SNP of the jth individual and pi is the frequency of the reference allele. SNPs were coded as minor allele dosage (0, 1, 2).

Next, by defining $g= W\mu$, $A= WW'/N$ and $\sigma g2 = N\sigma\mu 2$, Equation (2) is mathematically equivalent to Equation (3), which can be estimated by the restricted maximum likelihood approach.

$$Y = X\beta + g + \varepsilon, \text{ with Variance} = A\ \sigma g2+ I\varepsilon\sigma 2\varepsilon \tag{3}$$

where g is an n*1 vector of the total genetic effects of the individuals with $g \sim N(0, A\ \sigma g2)$, A is the genetic relationship matrix (GRM) between individuals and $\sigma 2g= N^*\sigma 2\mu$ is the total genetic variance explained by the SNPs. Hence $\sigma g2$ can be estimated by the restricted maximum likelihood approach, depending on the GRM estimated from the SNPs.

The gene-by-marriage interaction was assessed by comparing the proportion of variance explained—($\sigma 2g / (\sigma 2g +\sigma 2\varepsilon)$ in Equation (3)—between married and unmarried individuals. This form of G×E interaction is different from the traditional form of G×E in which a multiplicative interaction term is added in a regression. Conceptually, both the two forms of G×E examine the processes by which the effects of genes are conditioned by environmental factors or vice versa. In the traditional form, when modeling the interaction between marriage and 580 SNPs, it is most likely that one needs to either put 580 two-way interactions in a regression or run 580 regressions with each regression containing one two-way interaction. In our approach, 580 SNPs are simultaneously considered as random effects.

The proportion of variance explained was estimated for antisocial behavior at Waves III and IV separately. Specifically, we took the following steps to obtain the proportion of variance explained. First, the sample was divided into two groups—the married and the unmarried. Second, we performed subsample selection. Given that the sample consisted of siblings and twins, if related persons were included in the same mixed linear model the estimate of genetic effects would be biased by phenotypic correlations of, for example, siblings who shared common environments. Therefore we randomly selected an individual from every family. We did this separately for the married and the unmarried groups. Next, we repeated the subsample selection process 1,000 times to avoid the arbitrariness of which person in the family was selected. Finally, the mixed linear models were estimated for the married and unmarried groups separately and results were averaged over results obtained from 1,000 analytical subsamples. The Kolmogorov-Smirnov (KS) tests were conducted to compare the distribution of 1,000 proportions of variance explained between the married and the unmarried.

The Gene-Environment Correlation

Gene-environment correlations (rGEs) refer to situations in which genotypes are nonrandomly associated with environments. rGEs may bias estimates of G×E interactions (Wagner, Li, Liu, and Guo 2013). We tested whether the 580 SNPs were associated with marital status using the mixed linear model. The association was not significantly different from 0 ($p=0.96$). The evidence suggested that the rGE did not confound the G×E interaction results in this study.

**Results**

Table 2.2
The Effect of Marriage on Delinquency and Violence, Generalized Estimating Equations

| | Delinquency | Violence | Delinquency+violence |
|---|---|---|---|
| Married (ref: unmarried) | -0.17*** | -0.17*** | -0.34*** |
| Age | -0.19* | -0.09 | -0.27 |
| Age$^2$ | 0.00 | 0.00 | 0.00 |
| Female (ref: male) | -0.21*** | -0.43*** | -0.64*** |
| Race (ref: White) | | | |
| American Indian | 0.20 | -0.04 | 0.16 |
| Asian | -0.24* | -0.11 | -0.24* |
| Black | 0.09 | 0.08 | 0.00 |
| Multiracial | -0.05 | 0.03 | -0.02 |
| Other | -0.25 | -0.32* | -0.57** |
| Education (ref: no college) | | | |
| College or more | -0.01 | -0.15** | -0.15 |
| Employment (ref: unemployed) | | | |
| Employed | -0.07 | -0.20** | -0.26** |
| Church going (ref: less than weekly) | | | |
| Weekly or more | -0.06 | -0.10 | -0.17 |
| Household size (ref: 3-6) | | | |
| <3 | 0.03 | 0.04 | 0.07 |
| >6 | -0.09 | 0.02 | -0.07 |
| PVT score (ref: 90-110) | | | |
| <90 | -0.05 | 0.02 | -0.03 |
| >110 | 0.05 | -0.03 | 0.03 |
| Parental education (ref: high school) | | | |
| Below high school | 0.05 | -0.06 | -0.01 |
| More than high school | 0.06 | 0.05 | 0.11 |
| Closeness to parents (ref: close) | | | |
| Not close | 0.11 | 0.04 | 0.14 |
| | | | |
| Number of individuals | 1,254 | 1,254 | 1,254 |
| Number of observations | 2,364 | 2,369 | 2,367 |

*Note*: The dependent variables are delinquency and violence measured at Waves III and IV.
**p*<.05. ***p*<.01. ****p*<.001 (two-tailed tests).

Marriage and Antisocial Behavior

Table 2 presents the effect of marriage on delinquency and violence at Waves III and IV estimated in GEE models (Equation (1)). Married individuals showed a significant decrease on the delinquency and violence scales of 0.17 and a decrease of 0.34 on the sum of the two scales. This suggests that getting married would decrease the likelihood of behaving antisocially.

The Gene-by-Marriage Interaction

Results in Table 3 were obtained from the mixed linear models implemented in the GCTA software. Table 3 first presents the percent of variance in antisocial behavior explained by the 580 SNPs. Overall, the percent of variance explained was significantly smaller in married individuals than in unmarried individuals, suggesting that marriage may suppress the collective influence of the genes. Our hypothesis was supported. At Wave III, the SNPs jointly accounted for about 1.09%, 3.56% and 1.48% of the variance in delinquency, violence and the sum of delinquency and violence respectively in unmarried individuals, whereas the SNPs explained virtually no variance in the married. Similarly, at Wave IV the SNPs accounted for 0.26% and 0.14% of the variance in violence and the sum of delinquency and violence among the unmarried, and virtually none among the married. Variance explained by the SNPs can be seen as an estimator for heritability. We did not report results for delinquency at Wave IV because its distribution was highly right skewed.

Addressing Selection, the Confounding Effect of Age, and Population Heterogeneity

As mentioned previously, selection, age, and population heterogeneity may threaten the validity of the gene-by-marriage interaction findings. As for selection, we tested whether delinquent persons were less likely to get married. If earlier antisocial behavior at Waves I and II (1994-95 and 1996) were not a significant predictor for marital status at Waves III and IV (2001-02 and 2008-09), it suggests that selection based on antisocial behavior may not pose a serious threat to the deterrent capacity of marriage. Table 4 reports the results. The dependent variable, marital status, was a dichotomous variable with 1 indicating that a person is married and 0 otherwise. Logistic GEE models were used and the within-family correlations were addressed. None of the coefficients for delinquency, violence and the sum of delinquency and violence at Wave I or II were statistically significant. In other words, the probability of getting married was not associated with the levels of antisocial behavior earlier on.

Table 2.3
Percent of Variance in Delinquency and Violence Explained by 580 SNP, Mixed Linear Models Estimated in GCTA

| | Wave III dependent variable | | | | | | Wave IV dependent variable | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Delinquency | | Violence | | Delinquency+violence | | Violence | | Delinquency+violence | |
| | Married | Unmarried | Married | Unmarried | Married | Unmarried | Married | Unmarried | Married | Unmarried |
| Percent of variance explained by 580 SNPs | 0.00[a] | 1.09***[a] | 0.00[a] | 3.56***[a] | 0.00[a] | 1.48***[a] | 0.00[a] | 0.26***[a] | 0.09[a] | 0.14***[a] |
| Number of individuals | 191 | 835 | 193 | 837 | 191 | 837 | 428 | 546 | 428 | 546 |

*Note*: All models controlled for age, age squared, gender, race, education, employment, church going, household size, verbal IQ score, parental education, closeness to parents, and bio-ancestry scores. Because the distribution of delinquency at Wave IV was highly right skewed (over 90% of individuals scored 0 on the delinquency scale), estimates of the mixed linear models were not reliable. Therefore, we do not present results for delinquency at Wave IV.
[a]Kolmogrov-Smirnov test of whether the distribution of proportions of variance estimated in married individuals was smaller than in unmarried individuals.
**p*<.05. ***p*<.01. ****p*<.001.

To address the potential confounding effect of age, we first randomly excluded a subset of young unmarried individuals to generate a new sample so that the mean ages for married and unmarried individuals were the same. In our sample, the mean age for married and unmarried individuals was 26 and 24, respectively. In the new sample, the mean ages for the two groups were both 26. We call this new sample the age-comparable sample. A similar method has been used to equalize age in two groups in previous studies (e.g., Uggen 2000). Next, using this age-comparable sample analyses were carried out to examine whether marriage may suppress antisocial behavior in Equation (1), and whether marriage interacted with the genes in the mixed linear models. The left panel of Table 5 reports the results obtained from GEE models using the age-comparable sample. The marriage effect remained. Married individuals scored 0.23 less on delinquency, 0.20 less on violence and 0.43 on the sum of the two than unmarried individuals of comparable age. We also reestimated the mixed linear models in Table 3 using the age-comparable sample. The reestimation yielded similar results (not shown) to those presented in Table 3. Therefore, we are more confident in saying that age did not confound the marriage-antisocial-behavior association and the gene-by-marriage interaction results.

Table 2.4
Addressing Selection: Testing Whether More Antisocial Individuals Are Less Likely to Get Married by Using Delinquency and Violence at Waves I/II to Predict Marital Status at Waves III/IV, Generalized Estimating Equations

| | Married in 2001-02, Wave III | | | | | | Married in 2008-09, Wave IV | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) |
| Antisocial behavior in 1994-95, Wave I | | | | | | | | | | | | |
| Delinquency | 0.02<br>(0.62) | | | | | | -0.05<br>(0.19) | | | | | |
| Violence | | 0.05<br>(0.08) | | | | | | -0.02<br>(0.38) | | | | |
| Delinquency+violence | | | 0.03<br>(0.15) | | | | | | -0.02<br>(0.23) | | | |
| Antisocial behavior in 1996, Wave II | | | | | | | | | | | | |
| Delinquency | | | | 0.04<br>(0.50) | | | | | | -0.03<br>(0.41) | | |
| Violence | | | | | 0.02<br>(0.56) | | | | | | -0.02<br>(0.46) | |
| Delinquency+violence | | | | | | 0.02<br>(0.45) | | | | | | -0.02<br>(0.36) |
| Number of individuals | | 1,252 | | | 1,195 | | | 1,120 | | | 1,066 | |

*Note*: Numbers in parentheses are *p* values. All models controlled for age, gender, race, church going, household size, verbal IQ score, parental education, and closeness to parents. Results from (6×2 =) 12 regressions are presented as six measures of antisocial behavior at Waves I and II were used to predict marital status at Wave III or IV. Every entry is based on a separate regression.
**p*<0.05; ***p*<0.01; ****p*<0.001

In the right panel of Table 5, we explored the possibility that the deterrent capacity of marriage differed between persistent and nonpersistent offenders due to population heterogeneity. In our data, 55 respondents scored 1 or more on the violent behavior scale at all four waves and these 55 respondents were considered persistent offenders. The remaining individuals were coded as nonpersistent offenders. We added a dummy variable for the persistent offenders and an interaction between marriage and the dummy variable in GEE models. As expected, persistent offenders exhibited higher levels of delinquency and violence. But the interaction between marriage and persistent offender was not statistically significant. Therefore, no evidence supports the idea that marriage acted differently for persistent and nonpersistent offenders. In addition, excluding the 55 persistent offenders did not change results in Tables 2 or 3.

Table 2.5
Addressing the Confounding Effect of Age and Population Heterogeneity: Testing the Marriage Effect Using the Age-Comparable Sample, and Testing Whether the Marriage Effect Differs for Persistent Offenders, Generalized Estimating Equations

| | Age-comparable sample[a] | | | Original sample | | |
|---|---|---|---|---|---|---|
| | Delinquency | Violence | Delinquency +violence | Delinquency | Violence | Delinquency +violence |
| Married (ref: unmarried) | -0.23*** | -0.20*** | -0.43*** | -0.15*** | -0.13** | -0.28*** |
| Persistent offender (ref: nonpersistent offender) | ---- | ---- | ---- | 0.67** | 1.72*** | 2.39*** |
| Persistent offender × married | ---- | ---- | ---- | -0.16 | 0.37 | 0.18 |
| Number of individuals | 1,168 | 1,168 | 1,168 | 1,254 | 1,254 | 1,254 |
| Number of observations | 1,542 | 1,546 | 1,544 | 2,364 | 2,369 | 2,367 |

*Note*: The dependent variables are delinquency and violence measured at Waves III and IV. All models controlled for age, age squared, gender, race, education, employment, church going, household size, verbal IQ score, parental education, and closeness to parents.
[a]In the age-comparable sample, the mean ages were 26 for both married and unmarried individuals after randomly excluding a subset of young unmarried individuals. In the original sample, the mean age was 26 for married individuals and 24 for unmarried individuals. *$p$<0.05; **$p$<0.01; ***$p$<0.001

## Discussion

This study investigated whether marriage moderates the effects of 580 SNPs in 64 genes that are related to aggression and risky behavior on antisocial behavior. The main findings show that the SNPs explained much less of variance in delinquency and violence among married individuals than unmarried individuals, implying that marriage may suppress the collective genetic influence. Past inquiries about the effect of marriage on antisocial behavior primarily focus on the social, behavioral and psychological aspects. The integration of genetics enriches the theoretical frameworks. We found that marriage could work through a biological pathway—the modification of genetic effects—to deter delinquency and violence.

Moreover, evidence supports the inference that marriage caused declines in antisocial behavior, and therefore supports the validity of the gene-by-marriage interaction results. Selection, age, and population heterogeneity do not seem to pose serious threats. Our results are consistent with those of previous research that addressed causality in the marriage-crime nexus. This research showed that marriage may causally inhibit crime and deviant behavior using policy changes as natural experiments (Cáceres-Delpiano and Giolito 2008; Edlund, Yi, Li, and Zhang forthcoming), using statistical techniques such as propensity score matching and inverse probability of treatment weighting (King, Massoglia, and Macmillan 2007; Sampson, Laub, and Wimer 2006; Theobald and Farrington 2009), and taking advantage of a co-twin control design (Burt, Donnellan, Mikhila N. Humbad, Hicks, McGue, and Iacono 2010). With respect to population heterogeneity, we found that marriage did not influence differently for persistent and nonpersistent offenders. This pattern emerging from the sample of the general U.S. population is consistent with Blokland and Nieuwbeerta's (2005) finding based on a sample of the general Dutch population that the effect of marriage was the same for sporadic and low-rate offenders. Interestingly, in the same paper using data from a sample of Dutch criminal offenders Blokland and Nieuwbeerta found that the inhibiting effect of marriage only existed among low- and moderate-rate offenders, but not among high-rate offenders. Future work might examine why the marriage effect varies in different populations.

The gene-by-marriage interaction findings bear implications for researchers. High estimates of heritability for antisocial behavior from behavioral genetic studies (Rhee and Waldman 2002) may make it look as if environmental influences are not as important as genetic influences. Our findings pointed to the opposite. The effect of genes was conditional on the environment. Individuals possess different forms of genes related to antisocial behavior. Some individuals are more genetically susceptible to delinquency and violence than others. Regardless of the genotype, the collective influence of the genes is subject to the presence of marriage possibly because marriage can affect many aspects of an individual's life. Emotional attachment to the spouse, time devoted to the family, and normalized activity after marriage might all play a role in curbing the manifestation of the genes. Future research might investigate what aspects of marriage interact with genetic factors to deter antisocial behavior. In particular we found that the SNP explained approximately 1-3% of variance among unmarried individuals. The magnitude of variance explained in our findings was similar to that of genome wide association studies of complex traits such as body mass index (Speliotes, Willer, Berndt, and al 2010). In addition, our results suggest that sources of variation in delinquency and violence were more than the 64 genes and marriage. Other genes, epistasis, epigenetics, and gene expression may be associated with committing deviant behavior. Also, life events such as employment are worth investigation because they are turning points for desistance across the life course (Laub and Sampson 1993). More focused analyses of the roles of other biological pathways and life events would offer additional insights into the desistance process.

Several limitations should be acknowledged. We were unable to estimate the effects of genetic variants that were not covered by the SNP arrays. In addition, the 580 SNPs and causal alleles for delinquency and violence may not be in complete linkage disequilibrium. Therefore, the collective influence of the SNPs was likely to be underestimated. Also, this particular mixed linear model framework does not allow for analysis of genetically related individuals, resulting in reduction in sample size. Due to this our ability to investigate the roles of other factors in the desistance process was limited. For example, prior research suggests that gender contingencies are relevant to the marriage effect, and males tend to benefit more from the inhibiting influence of marriage (Giordano, Cernkovich, and Rudolph 2002; King, Massoglia, and Macmillan 2007; Sampson, Laub, and Wimer 2006). Future G×E research might consider using a larger sample to examine the roles of gender, race, and other factors.

Social scientists interested in G×E interaction are faced with two challenging tasks. The first is to identify "truly exogenous, causal environmental effects" (Conley 2009: 244). The second is to creatively use a variety of methods to detect G×E interactions (Shanahan and Boardman 2009). This article is just one example of how researchers may undertake these two tasks. Now SNP data is increasingly available in many large-scale social surveys. Rich datasets offer opportunities for future G×E research to employ different study designs and methods to gain a more comprehensive understanding of complex traits and behaviors.

## REFERENCES

Anholt, Robert R.H. and Trudy F.C. Mackay. 2012. "Genetics of Aggression." *Annual Review of Genetics* 46:145-164.

Beaver, Kevin M., John Paul Wright, Matt DeLisi, and Michael G. Vaughn. 2008. "Desistance from delinquency: The marriage effect revisited and extended." *Social Science Research* 37:736-752.

Blokland, Arjan A.J. and Paul Nieuwbeerta. 2005. "The Effects of Life Circumstances on Longitudinal Trajectories of Offending." *Criminology* 43:1203-1240.

Burt, S. Alexandra, M. Brent Donnellan, MA; Mikhila N. Humbad, Brian M. Hicks, Matt McGue, and William G. Iacono. 2010. "Does marriage inhibit antisocial behavior?: An examination of selection vs causation via a longitudinal twin design." *Archives of General Psychiatry* 67:1309-1315.

Cáceres-Delpiano, Julio and Eugenio Giolito. 2008. "The impact of unilateral divorce on crime." in *IZA Discussion Paper No. 3380*. Bonn, Germany: Institute for the Study of Labor.

Caspi, Avshalom, Joseph McClay, Terrie E. Moffitt, Jonathan Mill, Judy Martin, Ian W. Craig, Alan Taylor, and Richie Poulton. 2002. "Role of Genotype in the Cycle of Violence in Maltreated Children." *Science* 297:851-854.

Coccaro, Emil F., Richard J. Kavoussi, Robert L. Trestman, Steven M. Gabriel, Thomas B. Cooper, and Larry J. Siever. 1997. "Serotonin function in human subjects: intercorrelations among central 5-HT indices and aggressiveness." *Psychiatry Research* 73:1-14.

Conley, Dalton. 2009. "The Promise and Challenges of Incorporating Genetic Data into Longitudinal Social Science Surveys and Research." *Biodemography and Social Biology* 55:238-251.

Consortium, Rat Genome Sequencing Project. 2004. "Genome sequence of the Brown Norway rat yields insights into mammalian evolution." *Nature* 428.

Craig, Ian W. and Kelly E. Halton. 2009. "Genetics of human aggressive behaviour." *Human Genetics* 126:101-113.

DeLisi, Matthew. 2005. *Career Criminals in Society*: Sage.

Edlund, Lena, Junjian Yi, Hongbin Li, and Junsen Zhang. forthcoming. "Sex Ratios and Crime: Evidence from China." *The Review of Economics and Statistics*.

Farrington, David P. and Donald J. West. 1995. "Effects of Marriage, Separation, and Children on Offending by Adult Males." in *Current Perspectives on Aging and the Life Cycle, Vol.*

*14: Delinquency and Disrepute in the Life Course*, edited by Z. S. Blau and J. Hagan. Greenwich: JAI Press.

Giordano, Peggy C., Stephen A. Cernkovich, and Jennifer L. Rudolph. 2002. "Gender, Crime, and Desistance: Toward a Theory of Cognitive Transformation." *American Journal of Sociology* 107:990-1064.

Giordano, Peggy C., Ryan D. Schroeder, and Stephen A. Cernkovich. 2007. "Emotions and Crime over the Life Course: A Neo-Meadian Perspective on Criminal Continuity and Change." *American Journal of Sociology* 112:1603-1661.

Glueck, Sheldon and Eleanor Glueck. 1950. *Unraveling Juvenile Delinquency*. New York: The Commonwealth Fund.

Guo, Guang, Michael E. Roettger, and Tianji Cai. 2008. "The Integration of Genetic Propensities into Social-Control Models of Delinquency and Violence among Male Youths." *American Sociological Review* 73:543-568.

Harris, K.M., C.T. Halpern, E. Whitsel, J. Hussey, J. Tabor, P. Entzel, and J.R. Udry. 2009. "The National Longitudinal Study of Adolescent to Adult Health: Research Design."

Hennig, J., M. Reuter, P. Netter, C. Burk, and O. Landt. 2005. "Two Types of Aggression Are Differentially Related to Serotonergic Activity and the A779C TPH Polymorphism." *Behavioral Neuroscience* 119:16-25.

Hess, C., A. Reif, A. Strobel, A. Boreatti-Hümmer, M. Heine, K.-P. Lesch, and C. P. Jacob. 2009. "A functional dopamine-β-hydroxylase gene promoter polymorphism is associated with impulsive personality styles, but not with affective disorders." *Journal of Neural Transmission* 116:121-130.

Hirschi, Travis. 1969. *Causes of Delinquency*. Berkeley, CA: Free Press.

Hirschi, Travis and Michael Gottfredson. 1983. "Age and the Explanation of Crime." *American Journal of Sociology* 89:552-584.

Horney, Julie, D. Wayne Osgood, and Ineke Haen Marshall. 1995. "Criminal Careers in the Short-Term: Intra-Individual Variability in Crime and Its Relation to Local Life Circumstances." *American Sociological Review* 60:655-673.

King, Ryan D., Michael Massoglia, and Ross Macmillan. 2007. "The context of marriage and crime: Gender, the propensity to marry, and offending in early adulthood." *Criminology* 45:33-65.

Laub, J. H. and R. J. Sampson. 2001. "Understanding desistance from crime." Pp. 1-69 in *Crime and justice: A review of research*, vol. 28, edited by M. Tonry. Chicagao: University of Chicago Press.

Laub, John H., Daniel S. Nagin, and Robert J. Sampson. 1998. "Trajectories of Change in Criminal Offending: Good Marriages and the Desistance Process." *American Sociological Review* 63:225-238.

Laub, John H. and Robert J. Sampson. 2003. *Shared Beginnings, Divergent Lives: Delinquents Boys to Age 70*. Cambridge, MA: Harvard University Press.

Laub, John H. and Robert J. Sampson. 1993. "Turning Points in the Life Course: Why Change Matters to the Study of Crime." *Criminology* 31:301-325.

Linnoila, V. Markku and Matti Virkkunen. 1992. "Aggression, suicidality, and serotonin." *Journal of Clinical Psychiatry* 53:46-51.

Manucka, Stephen B., Janine D. Flory, Robert E. Ferrell, J.John Mann, and Matthew F. Muldoon. 2000. "A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity." *Psychiatry Research* 95:9-23.

Maxson, Stephen C. 2009. "The Genetics of Offensive Aggression in Mice." in *Handbook of Behavior Genetics*, edited by Y.-K. Kim. New York ;London: Springer.

McCarthy, Mark I., Gonçalo R. Abecasis, Lon R. Cardon, David B. Goldstein, Julian Little, John P. A. Ioannidis, and Joel N. Hirschhorn. 2008. "Genome-wide association studies for complex traits: consensus, uncertainty and challenges." *Nature Reviews Genetics* 9:356-369.

Miczek, Klaus A., Eric W. Fish, Joseph F. de Bold, and Rosa M. de Almeida. 2002. "Social and neural determinants of aggressive behavior: pharmacotherapeutic targets at serotonin, dopamine and γ-aminobutyric acid systems." *Psychopharmacology* 163:434-458.

Moffitt, Terrie E. 1993. "Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy." *Psychological Review* 100:674-701.

Mulvey, Edward P., Laurence Steinberg, Jeffrey Fagan, Elizabeth Cauffman, Alex R. Piquero, Laurie Chassin, George P. Knight, Robert Brame, Carol A. Schubert, Thomas Hecker, and Sandra H. Losoya. 2004. "Theory and Research on Desistance from Antisocial Activity among Serious Adolescent Offenders." *Youth Violence and Juvenile Justice* 2:213-236.

Murphy, Dennis L., Meredith A. Fox, Kiara R. Timpano, Pablo R. Moya, Renee Ren-Patterson, Anne M. Andrews, Andrew Holmes, Klaus-Peter Lesch, and Jens R. Wendland. 2008. "How the serotonin story is being rewritten by new gene-based discoveries principally related to SLC6A4, the serotonin transporter gene, which functions to influence all cellular serotonin systems." *Neuropharmacology* 55:932-960.

Nagin, Daniel and Raymond Paternoster. 2000. "Population Heterogeneity and State Dependence: State of the Evidence and Directions for Future Research." *Journal of Quantitative Criminology* 16:117-144.

Nelson, Randy J. and Brian C. Trainor. 2007. "Neural mechanisms of aggression." *Nature Reviews Neuroscience* 8:536-546.

Osgood, D. Wayne and Hyunkee Lee. 1993. "Leisure activities, age, and adult roles across the lifespan." *Society and Leisure* 16:181-208.

Osgood, D. Wayne, Janet K. Wilson, Patrick M. O'Malley, Jerald G. Bachman, and Lloyd D. Johnston. 1996. "Routine Activities and Individual Deviant Behavior." *American Sociological Review* 61:635-655.

Pritchard, J. K., M. Stephens, and P. Donnelly. 2000. "Inference of population structure using multilocus genotype data." *Genetics* 155:945-959.

Rhee, Soo Hyun and Irwin D. Waldman. 2002. "Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies." *Psychological Bulletin* 128:490-529.

Rubin, D. B. 1987. *Multiple Imputation for Nonresponse in Surveys*. New York: John Wiley and Sons.

Sampson, Robert J. and John H. Laub. 1993. *Crime in the Making: Pathways and Turning Points through Life*. Cambridge, MA: Harvard University Press.

Sampson, Robert J., John H. Laub, and Christopher Wimer. 2006. "Does marriage reduce crime? A counterfactual approach to within-individual causal effects." *Criminology* 44:465-508.

Shanahan, Michael J. and Jason D. Boardman. 2009. "Genetics and Behavior in the Life Course: A Promising Frontier." in *The Craft of Life Course Research*, edited by G. H. Elder and J. Z. Giele: The Guilford Press.

Shirtcliff, Elizabeth A., Douglas A. Granger, Alan Booth, and David Johnson. 2005. "Low salivary cortisol levels and externalizing behavior problems in youth." *Development and Psychopathology* 1:167-184.

Simons, Ronald L., Man Kit Lei, Steven R. H. Beach, Gene H. Brody, Robert A. Philibert, and Frederick X. Gibbons. 2011. "Social Environment, Genes, and Aggression : Evidence Supporting the Differential Susceptibility Perspective." *American Sociological Review* 76:883-912.

Soyka, M., U. W. Preuss, G. Koller, P. Zill, and B. Bondy. 2004. " Association of 5-HT1B receptor gene and antisocial behavior in alcoholism." *Journal of Neural Transmission* 111:101-109.

Speliotes, EK, CJ Willer, SI Berndt, and et al. 2010. "Association analyses of 249,796 individuals reveal 18 new loci associated with body mass index." *Nature Genetics* 42:937-948.

Statistics, Bureau of Justice. 2001-09. "Prison Inmates at Midyears 2001-09."

Theobald, Delphine and David P. Farrington. 2009. "Effects of Getting Married on Offending: Results from a Prospective Longitudinal Survey of Males." *European Journal of Criminology* 6:496-516.

Thornberry, Terence P. and Marvin D. Krohn. 2000. "The Self-Report Method for Measuring Delinquency and Crime." Pp. 33-83 in *Criminal Justice 2000: Innovations in Measurement and Analysis*, edited by D. Duffee, R. D. Crutchfield, S. Mastrofski, L. Mazerolle, D. McDowall, and B. Ostrom. Washington DC: ational Institute of Justice.

Uggen, Christopher. 2000. "Work as a Turning Point in the Life Course of Criminals: A Duration Model of Age, Employment, and Recidivism." *American Sociological Review* 65:529-546.

Wagner, Brandon, Jiang Li, Hexuan Liu, and Guang Guo. 2013. "Gene-Environment Correlation: Difficulties and a Natural Experiment-Based Strategy." *American Journal of Public Health* 103:S167-S173.

Waite, Linda J. 1995. "Does Marriage Matter?" *Demography* 32:483-507.

Warr, Mark. 1998. "Life-course transitions and desistance from crime." *Criminology* 36:183-216.

Yang, Jian, S. Hong Lee, Michael E. Goddard, and Peter M. Visscher. 2011. "GCTA: A Tool for Genome-wide Complex Trait Analysis." *American Journal of Human Genetics* 88:76-82.

# CHAPTER 3: GENDER AND RELIGIOSITY: RISK PREFERENCE THEORY REVISITED

## Introduction

The past two decades have seen a vigorous debate about whether the greater female religiousness can be explained by socialization, or by risk preference, or even by physiological differences between men and women (Collett and Lizardo 2009; Freese and Montgomery 2007; Miller and Hoffmann 1995; Miller and Stark 2002; Roth and Kroll 2007; Stark 2002; Sullins 2006). This debate attempts to solve a long-standing puzzle: why are women more religious than men?

It has been observed that women are more religious than men (Stark 2002; Walter and Davie 1998), and researchers have proposed various explanations. Some argue that greater religiousness among females may be the result of lower labor participation and higher demands from family (Iannaccone 1990; Vaus and McAllister 1987). Some argue that nurturing and submissive socialization for females may lead to higher prevalence of religious acceptance and commitment among females (Mol 1985; Suziedelis and Potvin 1981). Considering the lack in social power for women, some other argue that religion may be a compensation for the oppression women have in the social world (Turner 1991). However, there is little empirical evident supporting these socialization explanations.

Miller and colleagues propose risk preference theory to explain the gender gap in religiosity (Miller and Hoffmann 1995; Miller and Stark 2002). Risk preference theory argues that men are more likely to take risks and acceptance of religiousness can be viewed as risk-averse. Therefore, men are less religious than women. In addition, Stark (2002) argues that the gender gap is attributable to a physiological explanation. A debate emerges after the theory is proposed. Several articles evaluate this theory and the results do not lend support for risk preference explanations.

Given the important role of risk in the theory it is surprising that few studies in the debate have used direct measures of risk preference and biological risk (Freese and Montgomery 2007; Hoffmann 2009; Miller and Stark 2002). For example, indirect measures such as belief in afterlife (nonbelievers are considered as taking more risk) do not tap in risk aversion propensity well. Without proper measures, risk preference theory is still not fully tested and developed.

To shed new light on the old puzzle that why women are more religious, we move forward risk preference theory in three aspects. First, religiosity is a multidimensional concept but previous studies do not take this into account. We develop a framework for connecting different dimensions of religiosity with risk. Second, we test risk preference theory with direct measures of three types of risk—general, impulsivity, and sensation seeking risks, and discuss the relationship between the risks and religiosity. Third, we examine the role of genetic risk based on 41 genes related to aggression and offense to see whether gendered variation in religiousness has a genetic base.

**Risk Preference Theory**

Risk preference theory intends to “add one additional element to the pool of factors designed to explain why women are consistently more ‘religious’ than men” (Hoffmann 2009: 233). Miller and Hoffman (1995) argue that religiosity can be viewed in terms of risk. “One can conceive of religious acceptance as risk-averse behavior and the rejection of religious beliefs as risk-taking behavior” (Miller and Hoffmann 1995: 66). On the other hand, men are more likely to have a taste for risk than women in a variety of behaviors (e.g. Bromiley and Curley 1992;

Forthun, Bell, Peek, and Sun 1999; Hagan, Simpson, and Gillis 1988; Zuckerman, Ball, and Black 1990). Consequently, the gender gap in religiosity may be due to the gender difference in risk taking.

The idea that religiousness can be seen as risk aversion dates back to the Pascal's wager in the 17th century—there is nothing to lose by believing in God but potentially much to gain. However, believing in God may lose things. Gratifications need to be given up because many activities are prohibited by religion. In other words, risk takers who do not believe in God can have worldly gratifications that are considered sinful among the believers (Stark 2002). Miller and Hoffmann (1995) summarize that the risk analysis approach is consistent with many contemporary theories in sociology of religion. Religiosity may be viewed as a way of dealing with uncertainties in life (e.g., fear of death) (Malinowski 1925). Participation in religion may be a choice for risk-averse individuals to deal with uncertainties, but not for risk takers. From a rational choice perspective, the extent to which individuals decide to be religious depends on, partly, perceived risks and costs. Increase in perceived risks may lead to a rational decision to be more religious if the risks are associated with religion. To illustrate, Miller and Hoffmann give an example of parents who have children: "the perceived risk of not belonging to an institution that provides a moral education is likely to increase when a person has children." (Miller and Hoffmann 1995:65). Building on these arguments, Miller and Stark (2002) state that the reasons for why being irreligious can be seen as risk-taking are that "…religious doctrines specify serious consequences for irreligion… [and] [f]ailure to conform in terms of beliefs and practices, or the commission of 'sins,' can result in serious consequences, such as going to hell" (Miller and Stark 2002:1404).

Using data from a national sample of high school students in the United States, Miller and Hoffmann (1995) find that risk preference, which is measured by self-reported attraction to risk and danger, is negatively associated religiosity, and the gender gap in religiosity declines when risk preference is considered. Miller and Stark (2002) examine the gender gap within high-risk religions (e.g., among Christians, Muslims, and Orthodox Jews) and low-risk religions (e.g., among Buddhists and non-Orthodox Jews), using data from the World Value Surveys (WVS), General Social Survey (GSS) and the National Jewish Population Survey. The authors hypothesize that because within high-risk religions the consequences of being irreligious are expensive, larger gender differences are expected within such religion traditions. The findings support this hypothesis.

**The Debate**

A debate emerges after risk preference theory is proposed, partly because Miller and Stark (2002) argue that gender differences in religiousness are not caused by socialization. Using data from GSS, WVS and International Social Survey Program (ISSP), Sullins (2006) reports that social explanations are still important. The coefficient for gender declines substantially and model $R^2$ increases when social factors—socialization, structural location, and friends network–are introduced in the model. Collett and Lizardo (2009) examine whether mother's socioeconomic status, a proxy for gender-egalitarian socialization, is associated with the gender gap. The authors find that women raised by mothers with high socioeconomic status are less religious than women with low status mothers, and are closer to men in religiousness, using the GSS data. The authors conclude that gender-egalitarian values narrows gender differences in religiosity.

Some other studies involved in the debate indirectly evaluate risk preference theory by testing hypotheses derived from risk preference theory. Roth and Kroll (2007) argue that risk preference theory neglects the role of belief in an afterlife. The authors state that if risk preference theory is correct, then the following hypothesis is supposed to be supported: because risk perceptions only exist among those who believe in an afterlife, gender differences are larger among believers than nonbelievers. Using the GSS and WVS data, Roth and Kroll report findings that contradict this hypothesis. The authors' analysis shows that the gender gap in a set of religiosity measures is larger and more significant among nonbelievers than among believers, contrary to the predictions of risk preference theory. Freese and Montgomery (2007) test risk preference theory by testing a modified version of risk preference theory. The modified version takes into account not only afterlife punishments (believe in hell), but also afterlife rewards (believe in heaven). The authors demonstrate that one can get ambiguous predictions from this modified version of the theory. For example, among individuals who believe in heaven but not hell, males are predicted to be more religious than females whereas among individuals who believe in both heaven and hell predicted gender differences are expectedly small (Table 1 in the article). Using the WVS and ISSP data the authors report that the gender gap in religiosity is no smaller among those who do not believe in hell, which is the opposite of what risk preference theory predicts.

Although researchers in the debate acknowledge the potential importance of testing biological factors in risk preference theory, so far no study has done so. Stark (2002) posits that physiology (mainly testosterone level) can be responsible for risky behavior, and, if irreligiousness is to some extent a form of risky behavior, religiosity can be linked to physiology. Similarly, Miller and Stark (2002) conjecture that the gender gap in religiousness may be physiologically based. Some other claim that “[a] biological propensity to take risks should only be relevant for those who perceive a risk of posthumous punishment…” (Roth and Kroll 2007: 217). Sullins (2006) indirectly tests whether biological sex differences lead to the gender gap in religiosity. Sullins argues that if biological sex differences cause the gender gap, the gender gap is expected to appear universally regardless of social and/or cultural settings. Sullins’ analysis shows that men are more religious than women among Jews and Muslims. In this article, we directly test the role of genetic risk using molecular genetic data. We use 226 SNPs in 41 aggression-related genes to calculate genetic risk to test the relationship between religiosity and biological risk preferences.

**Varying Gender Gap in Religiosity**

Studies involved in the debate use a variety of variables to measure religiosity, but few pay much attention to the possibility that men and women perceive or construct religiousness in different ways (with an exception of Sullins (2006)). Miller and Stark (2002) report that the gamma correlation between gender and prayer is larger than the gamma correlation between gender and Bible authority, Bible reading, church attendance or denominational loyalty in the GSS data (Table 6 in their article). Roth and Kroll (2007) use the same five measures of religiosity in the GSS to examine the gender gap among nonbelivers and believers in life after death. Among nonbelievers the gender gap is found in prayer and biblical authority only. The authors, however, do not further interpret these findings as to why gender differences only appear in certain measures, nor do they discuss implications of the findings for gender differences in religiousness.

Examining the relationships among religious indicators, Sullin (2006) proposes a typology of “active” and “affective” religiousness. This typology is analogous to “the distinction between “spirituality” and “religiosity” in certain theological contexts, and to Luckman’s distinction between “implicit” and “explicit” religiousness as regards secularization (p. 849).” He argues that when expressing religiousness men are active, that is, oriented to action, and women are affective. His analysis of the GSS data shows that the standardized mean differences between men and women are larger for affective measures of religion such as frequency of prayer, felt closeness to God, and a self-assessment of religiousness than active measures of religion such as church membership, church attendance, and church activities. Freese and Montgomery (2007) note that researchers should concentrate on prayer to explain the gender difference in religious measures as they find that the bivariate differences in self-assessed religiousness and church attendance between men and women can be explained by the gender difference in prayer.

Gender variations between measures of religiosity are also reported and interpreted in previous studies. De Vaus and McAllister (1987) find that the differences between men and women in religious belief are larger than religious revelation, commitment or church attendance in Australia. Using a sample of respondents from the Akron area, Feltey and Poloma (1991) examine gender differences in six measures of religiosity—Orthodox, frequency of prayer, church attendance, encountered God during prayer, importance of religion, and intimacy with God. The regression results suggest that after controlling for gender ideology, which is measured by five Likert items asking about opinions about gender inequality, significant gender differences exist in prayer and intimacy with God, but not among the other measures. In both studies, the authors turn to socialization explanations for the gender gap. For example, De Vaus and McAllister posit that labor participation enables women to find a substitute for the benefits derived from religiousness, and as a result, the gender differences are smaller when labor participation is considered.

It is noteworthy that Feltey and Poloma (1991) state that the higher levels of intimacy with God among women can be partly attributed to a greater affectivity because women tend to be more nurturant and expressive than men in relationships. To illustrate, Feltey and Poloma uses Rubin's (1983) work. Rubin argues that the female sense of self is not as independent as the male sense of self. For women close personal relationships are "one of life's essential themes" (p 59), whereas for men interpersonal relationships are something they are not as good at because they are taught to "camouflage their feelings under cover" (p 71). Other personality factors may also account for gender differences in religiosity (Beit-Hallahmi and Argyle 1997). For example, researchers find that women have stronger guilt feelings and are more intro-punitive than men (Bernard 1949; Wright 1971). Hence, one possible explanation would be that individuals with guilt feelings are more religious as they seek forgiveness in religions.

To explain why gender differences in prayer and Bible reading are larger than other dimensions of religious life, Walter and Davie (1998) point out another possible explanation— "men engage in religious practices when they are publicly acceptable or even required, but tend not to bother with private devotions when there is no social pressure." In pre-industrial times, men, as the representative of the household, went to church and this was considered as a kind of public duty; whereas in modern times when religiousness becomes more of a private matter and optional social activity, men seem to drop away (McLeod 1981). In addition, based on statistics for membership of religious institutions and religious belief, Davie (1990) argues that in modern times belonging declines much faster than does believing, or modern religion can be described as "believing not belonging." Thus, the female religious motivations may fit better in contemporary religion than the male religious motivations.

## Genetic Origin of Risk Preference

Twin studies suggest that genetic influences account for approximately 20%-50% variation in various types of risk taking (e.g., Cesarini, Dawes, Johannesson, Lichtenstein, and Wallace 2009; Lin, Lyons, Scherrer, Griffith, True, Goldberg, and Tsuang 1998). Recent advances in genetics suggest that risk preference, a complex trait, is subject to the influence of a large number of genes; and numerous genetic variants have been associated with risk taking (Kreek, Nielsen, Butelman, and LaForge 2005). Genetic variants including Ankyrin repeat and kinase domain containing 1 (*ANKK1*), dopamine receptor D3 and D4 (*DRD3* and *DRD4*) and a serotonin transporter region (*5-HTTLPR*) are shown to be associated with risk taking and one form of risk preference—novelty seeking (Kreek, Nielsen, Butelman, and LaForge 2005; Kuhnen and Chiao 2009; Lusher, Chandler, and Ball 2001; Primus, Thurkauf, Xu, Yevich, Mcinerney, Shaw, Tallman, and Gallager 1997; Schinka, Letsch, and Crawford 2002). Genetic variations affect the physiology of the dopaminergic and serotonergic systems (CALDÚ and Dreher 2007; Hariri, Mattay, Tessitore, Kolachana, Fera, Goldman, Egan, and Weinberger 2002). So carriers of a particular form of the genotypes can be more likely to take risk. For example, individuals with two copies of the short allele of the *5-HTTLPR* take less risk than those with one or two copies of the long allele of the genotype (Cesarini et al. 2009).

## The Current Study

In this study, we move risk preference theory forward by directly testing whether three types of risk may explain the gender gap in religiosity. The three types of risk are general, impulsivity, and sensation seeking risk. With DNA data, we examine the role of genetic risk to test the physiological explanations for the gender gap. As discussed previously, there appears to be a gendered distinction between different dimensions of religiosity. We test whether different dimensions of religiosity are affected by risk in different ways, and whether risk can explain gender differences in all dimensions of religiosity. The present study focuses on the United States, rather than looking at cross national data.

**Data**

Data come from the genetic sample of the National Longitudinal Study of Adolescent Health (Add Health). Add Health started as a school-based study of adolescents in grades 7-12 in the United States (Harris, Halpern, Whitsel, Hussey, Tabor, Entzel, and Udry. 2009). A nationally representative sample of 80 schools was selected in 1994-95. In-home interviews collected data on approximately 20,000 respondents randomly chosen from the 80 schools. Respondents were interviewed in 1994-95 (Wave I) and followed up in 1995-96 (Wave II), 2001-02 (Wave III) and 2008-09 (Wave IV). During in-home interviews, respondents were asked about personality, religiosity and other information.

The genetic sample consists of full siblings, monozygotic twins and dizygotic twins. The total number of respondents in the genetic sample is 2,612. Saliva of the genetic sample was collected and genotyped at Wave III. Our genotyping is funded by a major National Science Foundation grant. DNA was isolated from buccal cells at the Institute of Behavior Genetics at the University of Colorado, Boulder. The average yield of DNA was 58 $\pm 1$ μg. The genotype data used in this analysis were based on an Illumina GoldenGate assay. The GoldenGate array targeted 1,536 SNPs including 186 ancestral informative markers. A total of 1,140 SNPs in 130 genes were successfully genotyped and survived cleaning. The number of respondents whose DNA was successfully genotyped was 2,281.

To calculate genetic risk scores, we select genes related to offense and aggression discovered in mouse studies. Mice are one of the ideal animals that can be studied to provide new knowledge for genetics of aggression in humans. Approximately 99% of genes in mice can find direct counterparts in humans (Consortium 2002). Humans and mice have similar neural pathways through which aggression is mediated (Nelson and Trainor 2007). Another major advantage of studying mice is that the phenotype of mice can be measured more precisely, and the genetic background and environmental conditions can be controlled more easily

Of 130  genes, 39 genes have been shown to be related to aggression and offense in transgenic or knockout studies of male and female mice (Maxson and Canastar 2003; Maxson 2009). We find that the *CCKBR* and *AVPR1A* genes in our genotype data have similar functions to those of the *CCK2* and *AVPR1B* genes summarized by Maxon (2009).  SNPs in these two genes are also included in the calculation of the genetic risk score. A total number of 41 autosomal genes are used in the analysis.

**Measures**

Religiosity is measured by five variables: belief in God, monthly frequency of prayer, monthly frequency of religious service attendance, how important religious faith is to the respondent, and how religious the respondent is. Belief in God is grouped into believing in God and otherwise. The original questions on prayer and attendance asked frequency in the past 12 months. We recode them into monthly frequency as follows. For prayer the recoding is never-0, less than once a month-0.5, once a month-1, a few time a month-2, once a week-4, a few times a week-10, once a day-30, and more than once a day-60, and for attendance the recoding is never-0, a few times-0.2, several times-0.5, once a month-1, 2 or 3 times a month-2.5, once a week-4 and more than once a week-9. Responses to the importance of religious faith are not important, somewhat important, very important and more important than anything else. Responses to the extent to which one is a religious person are not religious at all, slightly religious, moderately religious and very religious. These two variables are treated as ordinal. Frequency of prayer, self-accessed religiousness and attendance were asked at both Waves III and IV whereas belief in God and importance of religious faith were asked at Wave III only.

To make interpretation comparable, we code three types of risk so that higher values indicate higher levels of risk-aversion in all three risk measures. General risk is measured based on a question asked at Wave III “Do you agree or disagree that you like to take risks?” Responses to this question are strongly agree-1, agree-2, neither agree nor disagree-3, disagree-4 and strongly disagree-5. The value of general risk ranges from 1 to 5. The higher the value is, the more risk-aversion the respondent is.

To measure impulsive risk, we sum up responses to three questions on the Wave III questionnaire. “Do you agree or disagree that you go out of your way to avoid having to deal with problems in your life?” “Do you agree or disagree that when making a decision, you go with your ‘gut feeling’ and don’t think much about the consequences of each alternative?” and “Do you agree or disagree that you live your life without much thought for the future?” Responses to the questions are strongly agree-1, agree-2, neither agree nor disagree-3, disagree-4 and strongly disagree-5. The value of impulsive risk is in the range of 3 to 15 with higher values indicating lower risk taking propensities. Responses to the first question are reversely coded because the first question asks about risk aversion while the other two questions ask about risk taking. If there is more than one missing item, the impulsive risk propensity is coded as missing.

Sensation seeking risk is constructed based on a section of in-home interview at Wave III called “propensity for risk.” Among seven pairs of sentences, respondents are asked to choose one sentence that best describes her or him. These seven items come from the Sensation Seeking Scale (Zuckerman 1979). The Sensation Seeking Scale (SSS) is a widely used scale. It “specifically aims to predict the propensity to engage in risk-taking behavior that has an element of thrill, sensation, or disinhibition” (Meertens and Lion 2008: 1507). The SSS is a predictor of high risk activities (Zuckerman and Kuhlman 2000) and is associated with individual differences in cortical arousal thresholds and levels of enzymes and neurotransmitters affecting the central nervous system (Geen 1997).

The seven pairs of sentences are: (1) “I like wild, uninhibited parties” and “I like quiet parties with good conversation”; (2) “I often like to drink alcohol or smoke marijuana” and “I don’t like to drink alcohol or smoke, marijuana”; (3) “I like to have new and exciting experiences and sensations, even if they are a little frightening, unconventional, or illegal” and “I am not interested in experience for its own sake”; (4) “I like to date people who are physically exciting” and “I like to date people who share my values”; (5) “A person should have considerable sexual experience before marriage” and “It’s better if two married people begin their sexual experience with each other”; (6) “If I had lots of money, I would fly around the world and have fun like some rich people do” and “Even if I had the money, I would not want to just fly around the world and have fun like some rich people do”; (7) “I feel best after having a couple of drinks” and “Something is wrong with people who need liquor to feel good.” The risk aversion propensity is the sum of the seven items. The sentence that indicates risk taking is assigned a value of 1 and the other sentence that indicates risk aversion is assigned a value of 2. If there are more than three missing items, the risk aversion propensity is coded as missing.

Controls variables are age, education, employment status, income, marital status, whether the respondent has children, whether the respondent was raised in a religion, race and religion affiliation. Missing values in the controls are imputed by the multiple imputation technique (Rubin 1987). Buddhists, Jews, Hindus and Muslims are not included in the analysis due to small number of respondents in these religion affiliations. Table 1 reports brief definitions and descriptive statistics for variables used in the analysis.

Table 3.1
Description and Descriptive Statistics

| Variable | Description | Wave III | Wave IV |
|---|---|---|---|
| Belief in God (mean) | Believe in God and always have | 0.84 | -- |
| | 1-yes 0-no | | |
| Prayer (mean and S.D.) | Monthly frequency of private prayer when alone | 17.49 | 20.84 |
| | | (20.82) | (22.84) |
| Importance (percent) | "How important is your religious faith to you?" | | |
| | 3-more important than anything | 11 | 11 |
| | 2-very important | 43 | 43 |
| | 1-somewhat important | 33 | 33 |
| | 0- not important | 13 | 13 |
| self-assessed religiousness (percent) | "To what extent are you a religious person?" | | |
| | 3-very religious | 13 | -- |
| | 2-moderately religious | 35 | -- |
| | 1-slightly religious | 35 | -- |
| | 0-not religious at all | 17 | -- |
| Attendance (mean and S.D.) | Monthly frequency of religious service attendance | 1.66 | 1.60 |
| | | (2.52) | (2.50) |
| Gender (mean) | 1-female 0-male | 0.52 | 52 |
| General risk-aversion | | 2.55 | -- |
| (mean and S.D.) | | (1.06) | |
| Impulsive risk-aversion | | 9.75 | -- |
| (mean and S.D.) | | (1.84) | |
| Sensation seeking risk-avesion | | 10.83 | -- |
| (mean and S.D.) | | (2.14) | |
| Genetic eneral risk-aversion | | -0.51 | -- |
| (mean and S.D.) | | (0.75) | |
| | | (0.12) | |
| Genetic impulsive risk-aversion | | 0.91 | -- |
| (mean and S.D.) | | (2.76) | |
| Genetic sensation seeking risk-aversion | | -1.44 | -- |
| (mean and S.D.) | | (2.15) | |
| Age (mean and S.D.) | Age | 21.95 | 28.44 |
| | | (1.70) | (1.75) |
| Race (percent) | Asian | 7 | -- |
| | Black | 17 | -- |
| | Multiracial | 4 | -- |
| | American Indian | 2 | -- |
| | White | 68 | -- |
| | Other | 0.8 | -- |

| | | | |
|---|---|---|---|
| College (percent) | 1-college or higher 0-otherwise | 54 | 67 |
| Employed (percent) | Currently employed | | |
| | 1-yes 0-no | 71 | 81 |
| Income (percent) | <15k | 37 | 8 |
| | 15k-50k | 39 | 60 |
| | >50k | 25 | 32 |
| Married (percent) | Currently married | | |
| | 1-yes 0-no | 18 | 44 |
| Child (percent) | Has child | | |
| | 1-yes 0-no | 20 | 50 |
| Raised religious (percent) | Raised in religion | | |
| | 1-yes 0-no | 90 | -- |
| Religion (percent) | Black protestant | 5 | 5 |
| | Catholic | 25 | 25 |
| | Evangelical protestant | 32 | 31 |
| | Mainline protestant | 9 | 9 |
| | No religion | 19 | 19 |
| | Other religion | 10 | 11 |
| N | | 2,148-2,211 | 1,887-2,010 |

## Analytical Strategy

The dependent variables include belief in God (dichotomous), monthly frequencies of prayer (continuous), religious service attendance (continuous), importance of religious faith (ordinal) and how religious the respondent is (ordinal). To assess effects of the variables of interest on the religiosity measures, Generalized estimating equation (GEE) is used (Liang and Zeger 1986). GEE accommodates dichotomous, continuous and ordinal outcomes and accounts for correlations within repeated measures. Logistic GEE is employed for the dichotomous dependent variable, linear GEE for the continuous dependent variables and ordered logit GEE for the ordinal dependent variables. Exchangeable working correlation structure is specified to address the within-person and within-family correlations in GEE models. Equation (1) below describes the basic structure of the GEE model.

$$\text{Religiosity}_{ija} = \beta_0 + \beta_1\text{Female}_{ija} + \beta_2\text{RiskAversion}_{ija} + \beta_3\text{Controls}_{ija} \quad (1)$$

where Religiosity$_{ija}$ is the religiosity measure for individual $i$ from family $j$ at Wave $a$; $\beta_1$ is the coefficient for female (male is the reference group); $\beta_2$ is the coefficient for risk-aversion score; and $\beta_3$ represents coefficients for the controls. As noted above, belief in God and religious person are measured at Wave III only, whereas the remaining three religiosity outcomes are measured at both Waves III and IV. Hence when modeling belief in God and religious person, only data from Wave III are used.

Following principals of the polygenic score approach proposed by Purcell and colleagues (2009), we take four steps to calculate the genetic risk propensity score. First, we select the 41 autosomal genes and eliminate highly correlated SNPs (i.e., SNPs that are in linkage disequilibrium). After the pruning, 226 SNPs are available for analysis. Second, the sample is randomly split into a discovery subsample and a validation subsample. Third, in the discovery subsample we run random-effects models (to control for the correlations within family) to select SNPs that are associated with the self-reported risk at the 0.05 level of significance using 226 SNPs. Fourth, to calculate the genetic score, SNPs of individuals in the validation subsample are weighted by the coefficients obtained from random-effects models in the third step. For example, allele A is found to be associated with general risk in the discovery subsample and the coefficient for this risk allele A is 0.5. An individual in the validation subsample possesses two risk alleles, that is, two A's. This individual's genetic risk score for general risk is $0.5 \times 2 = 1$.

The purpose of randomly splitting the sample in the second step is to address false negative and false positive issues. Unlike rare Mendelian traits that are determined by a single gene or allele (Glazier, Nadeau, and Aitman 2002), risk taking and other complex traits are jointly influenced by a number of genetic effects (Kreek, Nielsen, Butelman, and LaForge 2005). This raises the issue of false negative—genetic variants with small effect may be unlikely to pass a stringent *p* value. To address this issue, Purcell and colleagues (2009) propose to calculate genetic score using liberal thresholds (*p* values from 0.1 to 0.5 in the discovery subsample). To reduce the likelihood of false positive results, we employ a more stringent p value, 0.05, in the discovery subsample.

When splitting the sample, a "leave one out" strategy is employed. That is, the sample is randomly split into five subgroups and each subgroup is treated as validation subsample in turn while the remaining four subgroups together are treated as discovery subsample. The sample is split by family ID so that genetically related individuals from the same family are always assigned to the same subsample. There are 1,428 families in the sample. On average each subgroup consists of 285.6 families. This "leave one out" strategy reduces the influence of potential relatedness between discovery and validation subsamples (Allen et al. 2010).

Regarding aggression-related genes found in mouse studies, Maxson (2009) points out that some genes affect male mice, some affect female mice, and some other genes affect both, suggesting there is an interaction effect between genes and sex. Therefore we add an interaction between SNP and sex to in random-effects model that selects risk alleles using the discovery subsample. Equation (2) describes the basic structure of random-effects model in the third step above:

$$\text{RiskAversionScore}_{ij} = \beta_0 + \beta_1\text{SNP}_{ij} + \beta_2\text{Female}_{ij} + \beta_3(\text{SNP}_{ij} \times \text{Female}_{ij}) + \beta_4\text{Bio-ancestryScores}_{ij} + e_j \qquad (2)$$

In equation (2) RiskAversionScore$_{ij}$ is the self-reported risk-aversion score for individual *i* from family *j*; $\beta_1$ is the coefficient for SNP; $\beta_2$ is the coefficient for female; $\beta_3$ is the coefficient for the interaction between SNP and female; $\beta_4$ is the coefficients for African and European ancestry scores for the purpose of addressing population stratification (McCarthy, Abecasis, Cardon, Goldstein, Little, Ioannidis, and Hirschhorn 2008); and $e_j$ is the unobserved effects at the family level. If the test of $\beta_1$ is significant (when Female = 0), $\beta_1$ is used to weight the risk allele among men in the validation subsample. If the joint test of $\beta_1$ and $\beta_3$ is significant (when Female = 1), $\beta_1$ and $\beta_3$ are used to weight the risk allele among women in the validation subsample. For example, if a woman possesses 2 risk alleles and $\beta_1$ is 0.1 and $\beta_3$ is 0.2, the genetic risk score for this woman is $0.1\times2\times1+0.2\times2\times1=0.6$. It is likely that genetic score for risk-aversion is negative as $\beta_1$ and/or $\beta_3$ could be negative or the sum of $\beta_1\text{SNP}_{ij}$ and $\beta_3(\text{SNP}_{ij}\times\text{Female}_{ij})$ could be negative. Because we are interested in the effect of one's genetic score relative to others' scores in the sample, negative values of genetic scores do not affect the estimates in the model.

**Results**

In Table 2 we test whether women are more risk-averse than men. Overall, there is a clear pattern that risk-aversion scores are all higher among women than men. The differences in general risk, impulsive risk and sensation seeking risk between women and men are 0.50, 0.34 and 1.00 respectively and the differences are all statistically significant. Similarly, the gender differences in three types of genetic risk are all statistically significant—0.23, 4.84 and 2.75 respectively for general risk, impulsive risk and sensation seeking risk.

Table 3.2
Gender differences in risk-aversion

| | General risk-aversion score | | Impulsive risk-aversion score | | Sensation seeking risk-aversion score | |
|---|---|---|---|---|---|---|
| | Female | Male | Female | Male | Female | Male |
| Self-reported score | | | | | | |
| Mean | 2.80 | 2.30 | 9.92 | 9.58 | 11.31 | 10.31 |
| Std | 1.08 | 0.97 | 1.80 | 1.87 | 2.04 | 2.11 |
| Max | 5 | 5 | 15 | 15 | 14 | 14 |
| Min | 1 | 1 | 3 | 3 | 4 | 4 |
| Genetic score | | | | | | |
| Mean | -0.40 | -0.63 | 3.25 | -1.59 | -0.12 | -2.87 |
| Std | 0.94 | 0.44 | 1.53 | 1.07 | 1.53 | 1.77 |
| Max | 2.40 | 0.69 | 7.62 | 0.75 | 3.65 | 1.39 |
| Min | -2.38 | -1.49 | -0.54 | -3.91 | -3.50 | -7.56 |
| N | 1143 | 1068 | 1143 | 1068 | 1143 | 1068 |

Table 3 presents coefficients and standard errors for gender and general risk. A positive coefficient indicates higher levels of religiousness. There are five sets of GEE models of religiosity in Table 3. The first set of models, labeled as Model 1, includes gender; the second set, labeled as Model 2, includes self-reported general risk risk-aversion score; Model 3 includes both gender and self-reported general risk risk-aversion score; Model 4 includes genetic general risk-aversion score; and the last set of models, labeled as Model 5, includes both gender and genetic general risk-aversion score. Models 1 to 5 all control for age, race, education, current employment status, income, marital status, whether was raised in a religion, whether has child, and religion affiliation. Model 1 shows that women are more religious than men on the five measures of religiosity—belief in God, prayer frequency, importance of religious faith, self-assessed religiousness and church attendance. Model 2 suggests that self-reported general risk is not associated with religiousness except for church attendance. Individuals with higher general risk-aversion score are more likely to go to church. In Model 3, for every measure of religiosity the coefficient for female remains statistically significant and the coefficient for self-reported general risk is not statistically significant, suggesting that general risk does not explain the gender gap. Similarly, Models 4 and 5 show that genetic general risk is not associated with any of the religiosity measures and cannot explain the gender gap either.

In Table 4, we report results based on impulsive risk. The structure of Table 4 is identical to that of Table 3. Models 1 to 5 in Table 4 are the same as those in Table 3 except that general risk is replaced by impulsive risk in the models. Women exhibit higher levels of religiosity, as suggested by Model 1. Model 2 shows that impulsive risk-aversion score is positively associated religiousness. In Model 3, results show that the inclusion of impulsive risk does not completely explain away sex differences in any measures of religiosity, although the magnitude of all the coefficients for female decreases. The genetic risk-aversion score are associated with religiosity in Model 4. But in Model 5 where gender and genetic impulsive risk are entered simultaneously, for three dependent variables, belief in God, importance of religious faith and self-assessed religiousness, the coefficients for gender and genetic risk become not significant and the standard errors increase substantially. This is most likely caused by multicollinearity—genetic impulsive risk is correlated with gender due to the interaction term in random-effects models that produces the genetic risk-aversion score. For the other two measures of religiosity—frequency of prayer and church attendance, the coefficient for gender remains statistically significant. Therefore, the results do not support the inference that the gender gap is explained by genetic impulsive risk.

Table 5 presents the effects of gender and sensation seeking risk on religiosity. Tables 5 and 3 share the same structure and models except that sensation seek risk replaces general risk in Table 3. Model 1 shows that women tend to be more religious. Model 2 shows that sensation seeking risk is associated with religiosity. The more risk-aversion a person is, the more religious this person is likely to be. The interesting finding of Model 3 is that the risk-aversion score explains away gender differences in self-assessed religiousness and church attendance—the coefficients in these three models turn into non-significant. However, gender differences in belief in God, prayer, and importance of religious faith remain statistically significant but are smaller than those in Model 1 where only gender is entered in the model. Model 4 shows that genetic sensation seeking risk-aversion has a positive effect on the level of religiousness. But, as Model 5 suggests, the gender gap cannot be explained by genetic sensation seeking risk although the magnitude of coefficients for female is smaller compared to Model 1.

Table 3.3

Estimates of the effects of gender/**general risk** on religiosity

| Dependent variable | Independent variable | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|---|
| Belief in God | Gender | | | | | |
| | Female | 0.42** | | 0.41** | | 0.40** |
| | | (0.13) | | (0.14) | | (0.13) |
| | Self-reported general risk | | | | | |
| | Risk-aversion score | | 0.06 | 0.02 | | |
| | | | (0.06) | (0.06) | | |
| | Genetic general risk | | | | | |
| | Risk-aversion score | | | | 0.10 | 0.06 |
| | | | | | (0.09) | (0.09) |
| | N | 2,184 | 2,184 | 2,184 | 2,184 | 2,184 |
| Prayer | Gender | | | | | |
| | Female | 5.80*** | | 5.84*** | | 5.96*** |
| | | (0.75) | | (0.78) | | (0.77) |
| | Self-reported general risk | | | | | |
| | Risk-aversion score | | 0.52 | -0.08 | | |
| | | | (0.36) | (0.37) | | |
| | Genetic general risk | | | | -0.14 | -0.71 |
| | Risk-aversion score | | | | (0.48) | (0.48) |
| | N | 4,212 | 4,212 | 4,212 | 4,212 | 4,212 |
| Importance | Gender | | | | | |
| | Female | 0.29*** | | 0.30*** | | 0.30*** |
| | | (0.07) | | (0.08) | | (0.07) |
| | Self-reported general risk | | | | | |
| | Risk-aversion score | | 0.01 | -0.02 | | |
| | | | (0.03) | (0.03) | | |
| | Genetic general risk | | | | | |
| | Risk-aversion score | | | | 0.01 | -0.02 |
| | | | | | (0.04) | (0.05) |
| | N | 4,210 | 4,210 | 4,210 | 4,210 | 4,210 |
| Self-assessed | Gender | | | | | |
| religiousness | Female | 0.25** | | 0.24** | | 0.25** |
| | | (0.09) | | (0.09) | | (0.09) |
| | Self-reported general risk | | | | | |
| | Risk-aversion score | | 0.06 | 0.03 | | |
| | | | (0.04) | (0.04) | | |
| | Genetic general risk | | | | | |
| | Risk-aversion score | | | | 0.04 | 0.01 |
| | | | | | (0.06) | (0.06) |
| | N | 2,199 | 2,199 | 2,199 | 2,199 | 2,199 |
| Attendance | Gender | | | | | |
| | Female | 0.29** | | 0.26** | | 0.30*** |
| | | (0.09) | | (0.09) | | (0.09) |
| | Self-reported general risk | | | | | |
| | Risk-aversion score | | 0.09* | 0.06 | | |
| | | | (0.04) | (0.04) | | |
| | Genetic general risk | | | | | |
| | Risk-aversion score | | | | -0.04 | -0.07 |
| | | | | | (0.06) | (0.06) |
| | N | 4,217 | 4,217 | 4,217 | 4,217 | 4,217 |

Notes: All 15 models in this table control for age, race, education, current employment status, income, marital status, whether was raised in a religion, whether has child, and religion affiliation in generalized estimating equations.

Standard errors in parentheses.

*p<0.05; **p<0.01; ***p<0.001 (two-tailed tests).

Table 3.4
Estimates of the effects of gender/**impulsive risk** on religiosity

| Dependent variable | Independent variable | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|---|
| Belief in God | Gender | | | | | |
| | Female | 0.42** | | 0.40** | | 0.13 |
| | | (0.13) | | (0.13) | | (0.29) |
| | Self-reported impulsive risk | | | | | |
| | Risk-aversion score | | 0.06# | 0.05 | | |
| | | | (0.03) | (0.04) | | |
| | Genetic impulsive risk | | | | | |
| | Risk-aversion score | | | | 0.08** | 0.06 |
| | | | | | (0.02) | (0.05) |
| | N | 2,183 | 2,183 | 2,183 | 2,183 | 2,183 |
| Prayer | Gender | | | | | |
| | Female | 5.82*** | | 5.63*** | | 8.31*** |
| | | (0.75) | | (0.75) | | (1.54) |
| | Self-reported impulsive risk | | | | | |
| | Risk-aversion score | | 0.78*** | 0.66*** | | |
| | | | (0.20) | (0.20) | | |
| | Genetic impulsive risk | | | | | |
| | Risk-aversion score | | | | 0.80*** | -0.52# |
| | | | | | (0.13) | (0.27) |
| | N | 4,211 | 4,211 | 4,211 | 4,211 | 4,211 |
| Importance | Gender | | | | | |
| | Female | 0.29*** | | 0.28*** | | 0.21 |
| | | (0.07) | | (0.07) | | (0.14) |
| | Self-reported impulsive risk | | | | | |
| | Risk-aversion score | | 0.04* | 0.04# | | |
| | | | (0.02) | (0.02) | | |
| | Genetic impulsive risk | | | | | |
| | Risk-aversion score | | | | 0.05*** | 0.02 |
| | | | | | (0.01) | (0.03) |
| | N | 4,209 | 4,209 | 4,209 | 4,209 | 4,209 |
| Self-assessed religiousness | Gender | | | | | |
| | Female | 0.25** | | 0.23** | | 0.19 |
| | | (0.09) | | (0.09) | | (0.17) |
| | Self-reported impulsive risk | | | | | |
| | Risk-aversion score | | 0.08*** | 0.07** | | |
| | | | (0.02) | (0.02) | | |
| | Genetic impulsive risk | | | | | |
| | Risk-aversion score | | | | 0.04** | 0.01 |
| | | | | | (0.02) | (0.03) |
| | N | 2,198 | 2,198 | 2,198 | 2,198 | 2,198 |
| Attendance | Gender | | | | | |
| | Female | 0.29** | | 0.27** | | 0.32# |
| | | (0.09) | | (0.09) | | (0.18) |
| | Self-reported impulsive risk | | | | | |
| | Risk-aversion score | | 0.07** | 0.06** | | |
| | | | (0.02) | (0.02) | | |
| | Genetic impulsive risk | | | | | |
| | Risk-aversion score | | | | 0.04** | -0.01 |
| | | | | | (0.02) | (0.03) |
| | N | 4,216 | 4,216 | 4,216 | 4,216 | 4,216 |

Notes: All 15 models in this table control for age, race, education, current employment status, income, marital status, whether was raised in a religion, whether has child, and religion affiliation in generalized estimating equations.
Standard errors in parentheses.
*p<0.05; **p<0.01; ***p<0.001 (two-tailed tests).

Table 3.5
Estimates of the effects of gender/**sensation seeking risk** on religiosity

| Dependent variable | Independent variable | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|---|
| Belief in God | Gender | | | | | |
| | Female | 0.40** | | 0.29* | | 0.34# |
| | | (0.14) | | (0.14) | | (0.18) |
| | Self-reported sensation risk | | | | | |
| | Risk-aversion score | | 0.12*** | 0.10*** | | |
| | | | (0.03) | (0.03) | | |
| | Genetic sensation risk | | | | | |
| | Risk-aversion score | | | | 0.07* | 0.02 |
| | | | | | (0.03) | (0.04) |
| | N | 2,155 | 2,155 | 2,155 | 2,155 | 2,155 |
| Prayer | Gender | | | | | |
| | Female | 5.68*** | | 4.29*** | | 5.10*** |
| | | (0.77) | | (0.78) | | (1.01) |
| | Self-reported sensation risk | | | | | |
| | Risk-aversion score | | 1.71*** | 1.48*** | | |
| | | | (0.18) | (0.18) | | |
| | Genetic sensation risk | | | | | |
| | Risk-aversion score | | | | 0.95*** | 0.21 |
| | | | | | (0.17) | (0.23) |
| | N | 4,076 | 4,076 | 4,076 | 4,076 | 4,076 |
| Importance | Gender | | | | | |
| | Female | 0.27*** | | 0.17* | | 0.23* |
| | | (0.07) | | (0.08) | | (0.09) |
| | Self-reported sensation risk | | | | | |
| | Risk-aversion score | | 0.13*** | 0.12*** | | |
| | | | (0.02) | (0.02) | | |
| | Genetic sensation risk | | | | | |
| | Risk-aversion score | | | | 0.05** | 0.02 |
| | | | | | (0.02) | (0.02) |
| | N | 4,076 | 4,076 | 4,076 | 4,076 | 4,076 |
| Self-assessed religiousness | Gender | | | | | |
| | Female | 0.25** | | 0.10 | | 0.20# |
| | | (0.09) | | (0.09) | | (0.11) |
| | Self-reported sensation risk | | | | | |
| | Risk-aversion score | | 0.17*** | 0.17*** | | |
| | | | (0.02) | (0.02) | | |
| | Genetic sensation risk | | | | | |
| | Risk-aversion score | | | | 0.05* | 0.02 |
| | | | | | (0.02) | (0.03) |
| | N | 2,129 | 2,129 | 2,129 | 2,129 | 2,129 |
| Attendance | Gender | | | | | |
| | Female | 0.26** | | 0.05 | | 0.25* |
| | | (0.09) | | (0.09) | | (0.11) |
| | Self-reported sensation risk | | | | | |
| | Risk-aversion score | | 0.23*** | 0.22*** | | |
| | | | (0.02) | (0.02) | | |
| | Genetic sensation risk | | | | | |
| | Risk-aversion score | | | | 0.04* | 0.01 |
| | | | | | (0.02) | (0.03) |
| | N | 4,081 | 4,081 | 4,081 | 4,081 | 4,081 |

Notes: All 15 models in this table control for age, race, education, current employment status, income, marital status, whether was raised in a religion, whether has child, and religion affiliation in generalized estimating equations.
Standard errors in parentheses.
*p<0.05; **p<0.01; ***p<0.001 (two-tailed tests).

## Conclusion and Discussion

In this article, using survey and genetic data from Add Health we test risk preference theory with direct measures of three types of risk. The findings show a more complicated picture than previously thought. Not all three types of risk are associated with religiosity. Our findings suggest that impulsive risk and sensation seek risk influence religiousness whereas general risk does not appear to be associated with most measures of religiosity except church attendance. As noted previously, serious consequences such as going to hell are assigned by religious doctrines. Individuals who do not accept religious obligations are risk takers in a sense. Impulsive risk and sensation seeking risk may both capture the nature of the concept of risk that is connected to religiosity. For example, if a person does not “consider much for the future” (one of the items in the measure of impulsive risk), this person’s probability of rejecting the idea that there are afterlife punishments is higher than those who think about and prepare for the future. Regarding general risk, it does not necessarily point to a particular type of risk. General risk may or may not imply specific risks associated with irreligiousness.

Interestingly, despite of the relative importance of sensation seeking risk and impulsive risk for religiosity, we find that the gender gap in two measures—self-assessed religiosity and church attendance—is only explained by sensation seeking risk but not impulsive risk. Compared to other types of risk, sensation seeking risk is more likely to tap into instant, worldly gratifications that are prohibited by religion doctrines such as drinking and sex before marriage. Impulsive risk may tap more into non-gratification types of risk including both worldly and afterlife risks. If such differences between the two types of risk are distinguished, the gender gap in self-assessed religiosity and church attendance may be largely due to that men tend to take risk in experiencing instant gratifications.

Our results show that sensation seeking risk and impulsive risk do not completely explain the gender gap in the other three dependent variables—belief in God, frequency of prayer and importance of religious faith. To some extent this aligns with prior findings that gender differences are larger in the affective dimension (e.g., church attendance) than in the active dimension of religiousness (e.g., prayer) (Sullins 2006). As discussed previously, researchers have offered reasons for the larger gender gap in the affective dimension including men's fear of intimacy with God and the religious motivations of women. These factors, together with the risk preferences, contribute to gender differences in the affective dimension of religiosity. Therefore, only part of the gender gap in the three measures of religiosity are explained—coefficients for gender in these measures shrink in magnitude after the risk is introduced in the models.

To test whether physiological differences between women and men are responsible for the gender gap in religiosity, we calculate genetic risk-aversion scores. We select genes that are related to aggression and offence in transgenic or knockout studies of mice, and use variants in these genes to obtain genetic scores. Furthermore, because different genes work differently among female and male mice (Maxson 2009), suggesting an interaction effect among mice as well as humans, we allow SNPs to interact with sex in the calculation of genetic scores. Our analysis provides evidence for the association between genetic sensation seeking risk and religiosity. However, there is no strong evidence that genetic general risk and impulsive risk influence religiosity. Genetic sensation seeking risk only accounts for part of variation in religiousness between women and men. No gender difference can be explained by genetic sensation seeking risk. Hence the results do not support the hypothesis that the gender gap in religiousness is physiologically based.

Although our work sheds some new light on the old puzzle that why women are more religious than men, the findings reveal the complexity of the relationship among gender, risk preferences and religiosity. For example, church attendance and belief in God represent two quite different dimensions of the concept of religiosity. The reasons for the gender gap in these two measures of religiosity can be quite different. Future research might conduct further investigates.

REFERENCES


Allen, Hana Lango. 2010. "Hundreds of variants clustered in genomic loci and biological pathways affect human height." *Nature* 467:832-838.

Beit-Hallahmi, Benjamin and Michael Argyle. 1997. *The Psychology of Religious Behaviour, Belief and Experience*. London: Routledge.

Bernard, J. 1949. "The Rosenzweig picture of frustration-study: I. Norms, reliability, and statistical evaluation." *Journal of Psychology* 28:325-332.

Bromiley, Philip and Shawn P. Curley. 1992. "Individual Differences in Risk Taking." in *Risk-taking Behavior*, edited by J. F. Yates. New York: John Wiley & Sons.

CALDÚ, Xavier and Jean-Claude Dreher. 2007. "Hormonal and Genetic Influences on Processing Reward and Social Information." *Annals of the New York Academy of Sciences* 1118:43-73.

Cesarini, David, Christopher T. Dawes, Magnus Johannesson, Paul Lichtenstein, and Bjoern Wallace. 2009. "Genetic variation in preferences for giving and risk-taking." *Quarterly Journal of Economics* 124:809-842.

Collett, Jessica L. and Omar Lizardo. 2009. "A Power-Control Theory of Gender and Religiosity." *Journal for the Scientific of Religion* 48:213-231.

Consortium, Mouse Genome Sequencing. 2002. "Initial sequencing and comparative analysis of the mouse genome." *Nature* 420:520-562.

Consortium, The International Schizophrenia. 2009. "Common polygenic variation contributes to risk of schizophrenia and bipolar disorder." *Nature* 460:748-752.

Davie, Grace. 1990. "Believing without Belonging: Is This the Future of Religion in Britain?" *Social Compass* 37:455-469.

Feltey, Kathryn M. and Margaret M. Poloma. 1991. "From Sex Differences to Gender Role Beliefs: Exploring Effects on Six Dimensions of Religiosity." *Sex Roles* 25:181-193.

Forthun, Larry F., Nancy J. Bell, Charles W. Peek, and Sheh-Wei Sun. 1999. "Religiosity, Sensation Seeking, and Alcohol/Drug Use in Denominational and Gender Contexts." *Journal of Drug Issues* 29:75-90.

Freese, Jeremy. 2004. "Risk Preferences and Gender Differences in Religiousness: Evidence from the World Values Survey." *Review of Religion Research* 46:88-91.

Freese, Jeremy and James Montgomery. 2007. "The Devil Made Her Do It: Evaluating Risk Preference as an Explanation of Sex Differences in Religiousness." Pp. 187-230 in

*Advances in Group Processes: The Social Psychology of Gender*, edited by S. J. Correll. Oxford: Elsevier.

Geen, R. G. 1997. "Psychophysiological approaches to personality." in *Handbook of Personality Psychology*, edited by J. J. Hogan and S. Briggs. London: Academics Press.

Glazier, Anne M., Joseph H. Nadeau, and Timothy J. Aitman. 2002. "Finding Genes That Underlie Complex Traits." *Science* 298:2345-2349.

Hagan, John, John Simpson, and A. R. Gillis. 1988. "Feminist Scholarship, Relational and Instrumental Control, and a Power-control Theory of Gender and Delinquency." *British Journal of Sociology* 39:301-336.

Hariri, Ahmad R., Venkata S. Mattay, Alessandro Tessitore, Bhaskar Kolachana, Francesco Fera, David Goldman, Michael F. Egan, and Daniel R. Weinberger. 2002. "Serotonin Transporter Genetic Variation and the Response of the Human Amygdala." *Science* 297:400-403.

Harris, K.M., C.T. Halpern, E. Whitsel, J. Hussey, J. Tabor, P. Entzel, and J.R. Udry. 2009. "The National Longitudinal Study of Adolescent Health: Research Design." vol. 2014.

Hoffmann, John P. 2009. "Gender, Risk, and Religiousness: Can Power Control Provide the Theory?" *Journal for the Scientific of Religion* 48:232-240.

Iannaccone, Laurence R. 1990. "Religious Practice: A Human Capital Approach." *Journal for the Scientific of Religion* 29:297-314.

Kreek, Mary Jeanne, David A. Nielsen, Eduardo R. Butelman, and K. Steven LaForge. 2005. "Genetic Influences on Impulsivity, Risk Taking, Stress Responsivity and Vulnerability to Drug Abuse an Addiction." *Nature Neuroscience* 8:1450-1457.

Kuhnen, Camelia M. and Joan Y. Chiao. 2009. "Genetic Determinants of Financial Risk Taking." *Plos ONE* 4:e4362.

Liang, Kung-Yee and Scott L. Zeger. 1986. "Longitudinal data analysis using generalized linear models." *Biometrika* 73:13-22.

Lin, Seth A. Eisen Nong, Michael J. Lyons, Jeffrey F. Scherrer, Kristin Griffith, William R. True, Jack Goldberg, and Ming T. Tsuang. 1998. "Familial influences on gambling behavior: an analysis of 3359 twin pairs." *Addiction* 93:1375-1384.

Lusher, J. M., C. Chandler, and D. Ball. 2001. "Dopamine D4 receptor gene (DRD4) is associated with Novelty Seeking (NS) and substance abuse." *Molecular Psychiatry* 6:497-499.

Malinowski, Bronislaw. 1925. "Magic, science, and religion." Pp. 18-94 in *Science, religion and reality*, edited by J. Needham. New York: Macmillan.

Maxson, S. C. and A. Canastar. 2003. "Conceptual and methodological issues in the genetics of mouse agonistic behavior." *Hormones and Behavior* 44:258-262.

Maxson, Stephen C. 2009. "The Genetics of Offensive Aggression in Mice." in *Handbook of Behavior Genetics*, edited by Y.-K. Kim. New York ;London: Springer.

McCarthy, Mark I., Gonçalo R. Abecasis, Lon R. Cardon, David B. Goldstein, Julian Little, John P. A. Ioannidis, and Joel N. Hirschhorn. 2008. "Genome-wide association studies for complex traits: consensus, uncertainty and challenges." *Nature Reviews Genetics* 9:356-369.

McLeod, H. 1981. *Religion and the People of Western Europe*. Oxford: Oxford University Press.

Meertens, Ree M. and Rene Lion. 2008. "Measuring an Individual's Tendency to Take Risks: The Risk Propensity Scale." *Journal of Applied Social Psychology* 38:1506-1520.

Miller, Alan S. and John P. Hoffmann. 1995. "Risk and Religion: An Explanation of Gender Differences in Religiosity." *Journal for the Scientific of Religion* 34:63-75.

Miller, Alan S. and Rodney Stark. 2002. "Gender and Religiousness: Can Socialization Explanations Be Saved?" *American Journal of Sociology* 107:1399-1423.

Mol, Hans. 1985. *The Faith of Australians*. Sydney: George, Allen, & Unwin.

Nelson, Randy J. and Brian C. Trainor. 2007. "Neural mechanisms of aggression." *Nature Reviews Neuroscience* 8:536-546.

Primus, Renee J., Andrew Thurkauf, Jing Xu, Eileen Yevich, Sean Mcinerney, Kenneth Shaw, John F. Tallman, and Dorothy W. Gallager. 1997. "Localization and Characterization of Dopamine D4 Binding Sites in Rat and Human Brain by Use of the Novel, D4 Receptor-Selective Ligand [3H]NGD 94-1." *Journal of Pharmacology and Experimental Therapeutics* 282:1020-1027.

Roth, Louise Marie and Jeffrey C. Kroll. 2007. "Risky Business: Assessing Risk Preference Explanations for Gender Differences in Religiosity." *American Sociological Review* 72:205-220.

Rubin, D. B. 1987. *Multiple Imputation for Nonresponse in Surveys*. New York: John Wiley and Sons.

Rubin, L. 1983. *Intimate Strangers*. New York: Harper & Row.

Schinka, J.A., E.A. Letsch, and F.C. Crawford. 2002. "DRD4 and novelty seeking: Results of meta-analyses." *American Journal of Medical Genetics* 114:643-648.

Stark, Rodney. 2002. "Physiology and Faith: Addressing the "Universal" Gender Difference in Religious." *Journal for the Scientific of Religion* 41:495-507.

Sullins, D. Paul. 2006. "Deconstructing Universality, Constructing Complexity." *American Journal of Sociology* 112:838-880.

Suziedelis, Antanas and Raymond H. Potvin. 1981. "Sex Differences in Factors Affecting Religiousness among Catholic Adolescents." *Journal for the Scientific of Religion* 20:38-50.

Turner, Bryan. 1991. *Religion and Social Theory*. London: Sage Publications.

Vaus, David de and Ian McAllister. 1987. "Gender Differences in Religion: A Test of the Structural Location Theory." *American Sociological Review* 52:472-481.

Walter, Tony and Grace Davie. 1998. "The Religion of Women in the Modern West." *British Journal of Sociology* 49:640-660.

Wright, D. 1971. *The Psychology of Moral Behavior*. Harmondsworth: Penguin.

Zuckerman, Marvin. 1979. *Sensation seeking: Beyond the optimal level of arousal*. Hillsdale, NJ: Lawrence Erlbaum.

Zuckerman, Marvin, Samuel Ball, and James Black. 1990. "Influences of Sensation Seeking, Gender, Risk Appraisal, and Situational Motivation on Smoking." *Addictive Behavior* 15:209-220.

Zuckerman, Marvin and D. M. Kuhlman. 2000. "Personality and Risk-taking: Common Biosocial Factors." *Journal of Personality* 68:999-1029.